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1. Spencer, H.; Berger, E.; Charles, M.; Gottesman, E. D., and Laszlo, D.: Effects of 17-alpha-Ethyl-17-Hydroxy-Norandrostenone. Proc. Am. A. for Cancer Res. *2*:149 (April) 1956.

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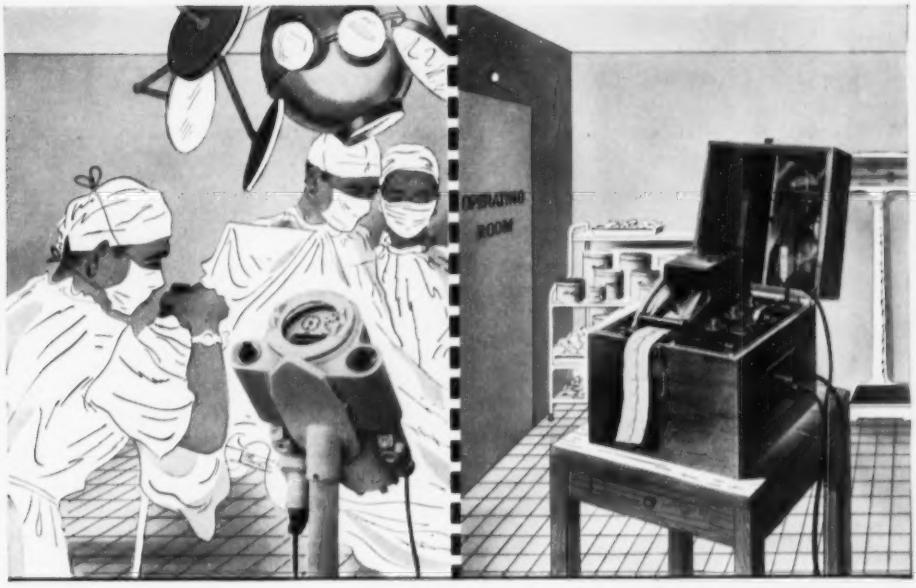

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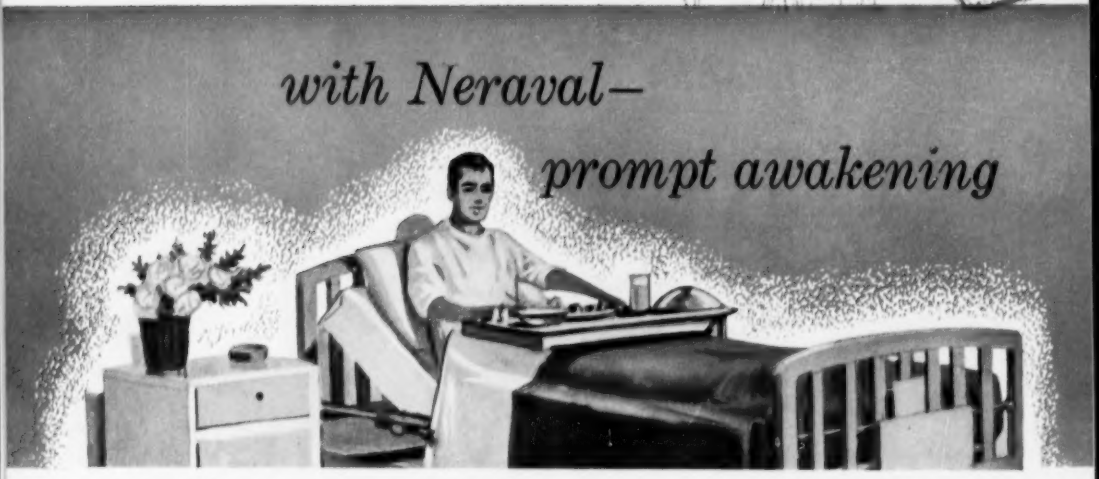

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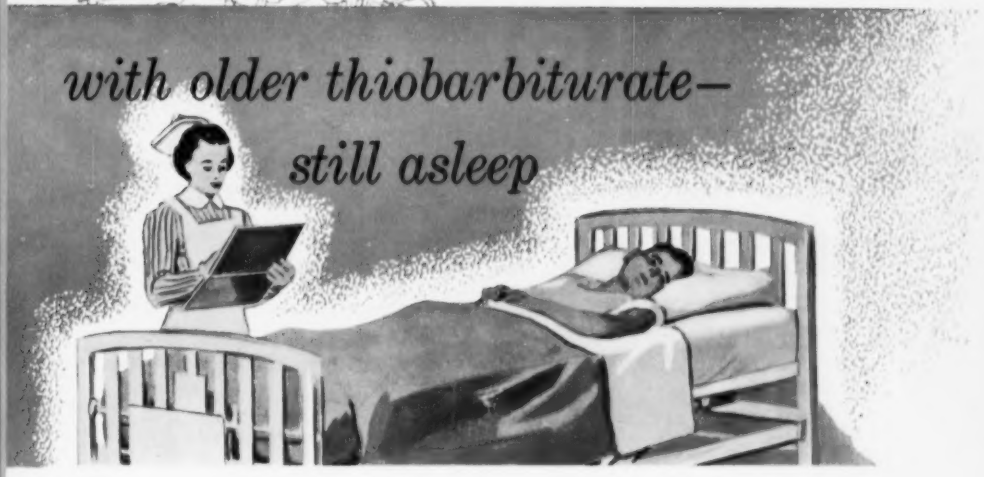

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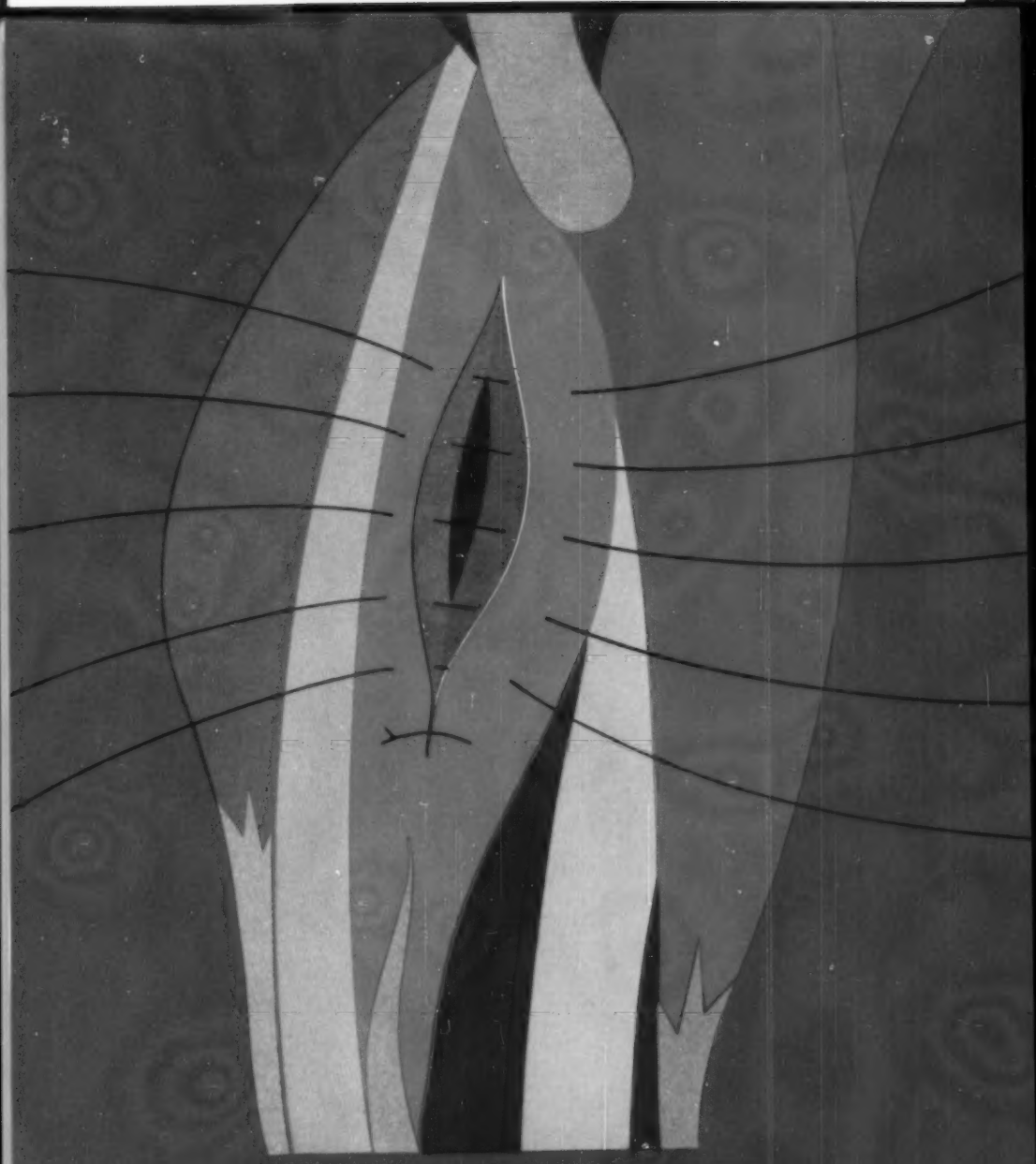

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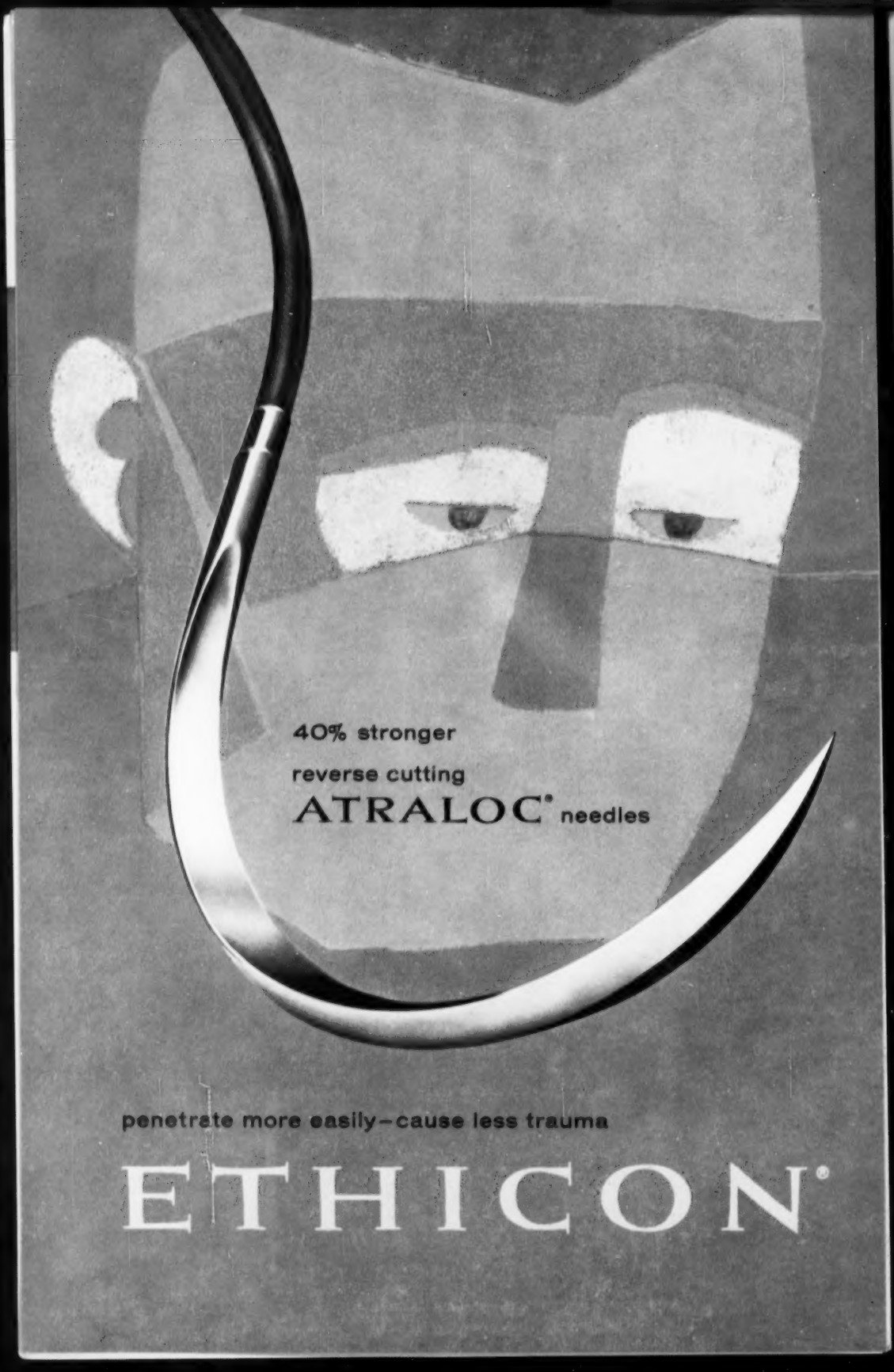
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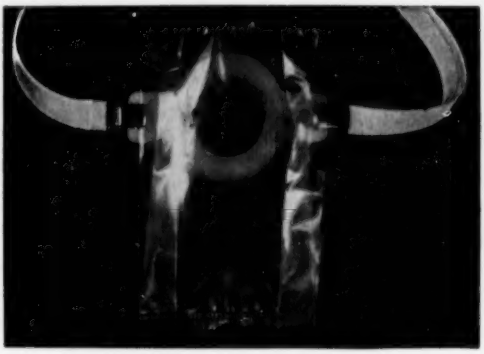

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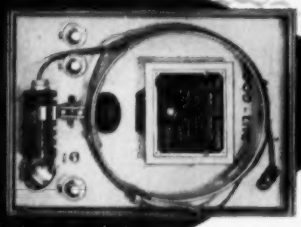

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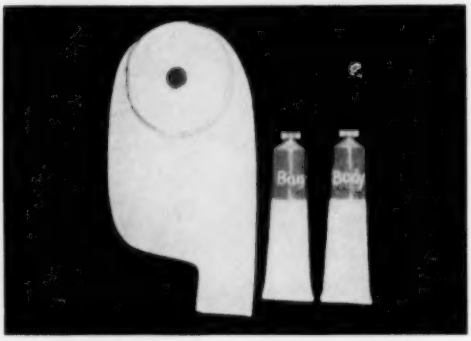

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Thoracoabdominal Aneurysm Involving Celiac, Superior Mesenteric, and Renal Arteries

Report of a Case Successfully Treated by Resection and Nylon-Graft Replacement

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Aneurysm of the thoracoabdominal aorta involving the celiac, superior mesenteric, and both renal arteries is a relatively uncommon entity. Not until recently have surgical attempts at altering the grave prognosis of such lesions been made with real hope of success. A survey of the literature reveals five patients who have undergone successful resection of aneurysms of this extent.[1,2] The present case is reported because it is believed to be the sixth documented instance of a patient surviving such a procedure and the first in which a nylon-graft replacement has been used.

Report of Case

This 41-year-old Negro man was admitted via the accident ward of University Hospitals on Jan. 5, 1957, with the chief complaint of severe lumbar back pain, which was constant, throbbing in nature, and came on suddenly during a bowel movement. He gave a history of recurrent episodes of lumbar backache of moderate severity, exaggerated by physical activity, during the six weeks prior to admission. It was known that he had undergone an appendectomy in September, 1956, through a McBurney incision. At that time the right iliac artery and aortic bifurcation were explored because of a diminished right femoral pulse which had been noted on physical examination prior to surgery. The right iliac artery was described as being smaller than normal, but the aortic bifurcation appeared normal, without evidence of aneurysm or occlusion.

At the time of the present admission, physical examination revealed that the blood pressure was 170/90, pulse 80, temperature 36 C (96.8 F), respirations 20. He was a well-developed, well-nourished man, in moderate distress from severe lumbar pain. The heart was not enlarged, and there was a regular sinus rhythm. No murmurs were heard. The chest was clear to percussion and auscultation. The diaphragms appeared to be moving normally. The abdomen was not distended, but there was moderately severe guarding bilaterally and some spasm of the abdominal musculature. When moderate pressure was placed upon the mid-abdomen, the pain in the back was made severer. There were hypoactive bowel sounds and a mass palpable in the left epigastrium, which appeared to be pulsating. There was an estimated 50% reduction in the volume of the right femoral pulse. A systolic murmur was heard over both femoral arteries and in the midline of the abdomen. The extremities were not remarkable except for the peripheral pulses, which were decreased in the right leg as compared with the left. The patient was admitted with a working diagnosis of aneurysm of the abdominal aorta. The laboratory findings were within normal limits. X-rays on admission revealed a shadow in the midline consistent with an aneurysm. Intravenous pyelograms revealed lateral displacement and slight anterior rotation of both kidneys. The information to be gained from an emergency aortogram was not thought to justify the risk of inserting a needle into the aneurysm. From a review of the patient's history and physical examination, it was believed that the most likely diagnosis was an aortic aneurysm which was either dissecting or leaking. After his admission to the

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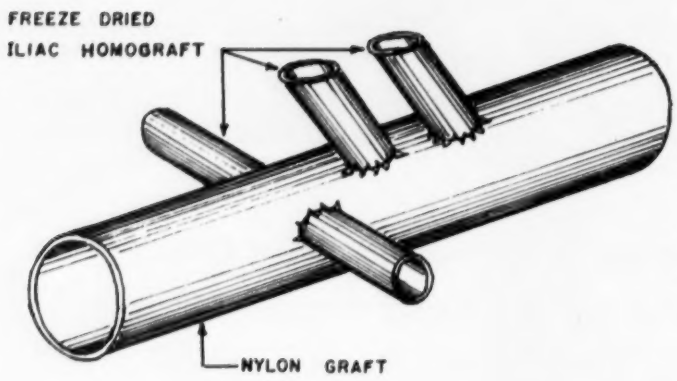

Fig. 1.—Drawing of the prosthesis used in the case presented. The branches shown were sutured to the celiac, superior mesenteric, and both renal arteries.

hospital, the patient's vital signs remained stable for 12 hours. However, the lumbar back pain continued to radiate upward and was unrelieved by narcotics. These developments seemed to substantiate the diagnosis of dissecting aneurysm, and the patient was taken to surgery.

With the patient under satisfactory endotracheal anesthesia, an abdominal midline incision was made, starting below the umbilicus and extending upward into the eighth intercostal space. When the abdomen was entered, a large aneurysm could be felt beneath the pancreas and stomach. The aorta was exposed by dividing the diaphragm and was dissected free above the aneurysm. A tape was then passed around the aorta so that hemorrhage could be controlled. The spleen was removed, and the colon, stomach, and pancreas were reflected to the right side of the abdomen after dividing the peritoneal attachments, thus exposing the aneurysm. The aneurysm was then dissected free of surrounding structures and was found to begin just above the diaphragm and to involve the celiac axis, the superior mesenteric artery, and both renal arteries. It ended just above the inferior mesenteric artery. The lower portion of the abdominal aorta was then dissected free and a controlling tape placed about it at this point. Both renal arteries, the superior mesenteric artery, and the celiac axis were dissected free of all surrounding tissue to provide adequate arterial length for suture. The inferior vena cava was then dissected from the right side of the aneurysm and the aneurysm completely isolated of all surrounding structures.

A crimped nylon by-pass was then sutured by end-to-side anastomosis into the normal aorta above and below the points at which the occluding clamps would be placed. This allowed a free by-pass of blood to the distal aorta at the time the aorta was occluded during resection of the aneurysm.

A frozen-dried homograft was not available to replace the aneurysm which was to be removed; so a graft was prepared by using a large woven nylon graft and suturing to it stumps of frozen-dried homograft from iliac arteries (Fig. 1). These stumps for the celiac axis, the superior mesenteric artery, and both renal arteries were sutured in the appropriate places to provide ample length for the anastomoses. The aorta was then clamped above and below the site of the aneurysm. The aorta and the branching vessels were transected, and the aneurysm was removed. The graft was then placed in the defect, and the two aortic ends were anastomosed. Originally, it had been planned to suture first the lower anastomosis and then a renal artery. Removal of the lower clamps would then allow blood to flow to one kidney through the by-pass. However, it was noted that the by-pass was not working very well and that adequate renal blood flow would probably not be reestablished by this method. For this reason, the upper and lower aortic anastomoses were made, and then the left renal artery was anastomosed. The clamps were removed, and blood was allowed to flow to the left kidney. The left kidney was without blood flow for approximately 53 minutes. As rapidly as possible, the right renal artery was then anastomosed, and blood was flowing to the right kidney at 1 hour and 10 minutes. The superior mesenteric artery was next anastomosed and blood was flowing through it at 1 hour and 40 minutes, and, finally, the celiac axis, as the last stage of the procedure, was anastomosed and its blood flow reestablished after two hours.

At the time of transecting the aorta below the aneurysm, it was noted that there was a false channel which had dissected down the aorta and was the apparent cause of the diminished right femoral pulse. The lower anastomosis was made with the true lumen, the false passage having been obliterated. Once the graft was in place and all hemorrhage was controlled, the colon, pancreas, and stomach were sutured in place and the raw surfaces were reperitonealized. The diaphragm was then closed with interrupted sutures. The ab-

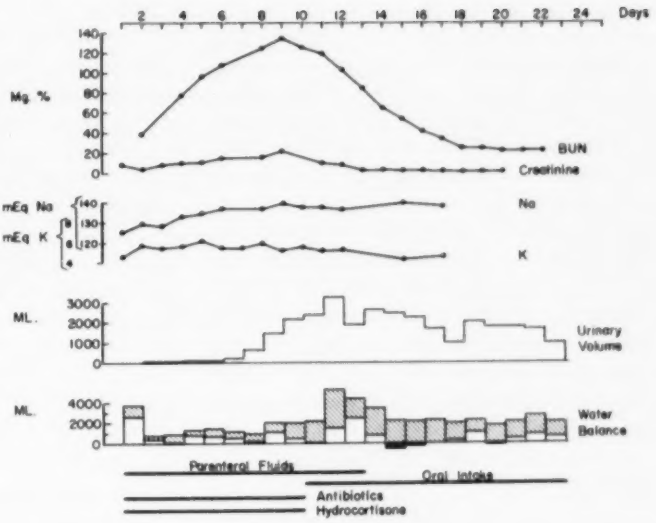

Fig. 2.—Data reflecting the metabolic changes during recovery. The water balance is plotted as follows: The intake is plotted upward from the horizontal zero line, and the output, from the top of the intake column downward. A positive balance is shown by the clear white area above the zero line, whereas extension of the column below this line represents a negative balance.

dominal incision was closed with through-and-through stainless steel wire, and the chest incision was closed in layers. A posterior thorocotomy tube was inserted and placed on underwater drainage. Because of the prolonged anesthesia with an endotracheal tube and because of the extent of the incision, it was felt that the patient's respiratory problems would be critical and would be one of the controlling factors in survival. Therefore, an elective tracheotomy was accomplished at the end of the procedure. The patient tolerated the procedure well, the total operating time being approximately 12 hours and 23 units of blood being required. Three of these units were given as freshly drawn whole blood in plastic bags in order to provide adequate clotting for the patient, such large amounts of banked blood having been given.

Postoperatively he was placed in an oxygen tent with humidity, and a nasogastric tube was passed and placed on constant suction to prevent gastric dilatation. Hydrocortisone, 100 mg. per day, was given for two reasons. 1. Part of the blood supply to each adrenal gland had been interrupted when the aneurysm was removed, and, although survival of the adrenals was expected, it was felt that adrenal function might not be adequate for the first few days. 2. The left adrenal gland was exposed to the air during most of the procedure and had sustained a certain amount of trauma, so that its state of function was questionable. The hydrocortisone dose was tapered off at the end of six days.

The patient was given fluids, as shown in Figure 2. The urinary output in the first 24 hours after surgery totaled 48 cc., and a diagnosis of acute tubular necrosis was made. Because of this, fluid restriction was instituted immediately, the daily intake being limited to 600-700 cc. plus the amount of any renal or extrarenal losses that could be measured. Figure 2 also illustrates the blood chemical and other significant findings. His temperature rose to a maximum of 38.5 C (101.3 F) on the third postoperative day, then gradually fell to normal, and his urinary output began to rise on the sixth hospital day and continued to increase over the following two weeks. Bowel sounds returned on the seventh postoperative day, and oral fluids were started on the ninth postoperative day. The blood urea nitrogen rose to 126 mg. per 100 cc. on the ninth postoperative day and then began to fall, and had returned to normal by the 25th postoperative day. Serum potassium never rose above 6.1 mEq. per liter. Bowel function returned to normal, as did his appetite. He was discharged on the 30th postoperative day feeling well, although still having occasional bouts of pain in the thoracic incision.

On last follow-up examination, at five months, the patient was found to have gained weight and was feeling well. All pulses in the periphery were strong and bilaterally equal, and it must be assumed that the internal anastomoses were working properly.

Comment

When this patient was admitted to the hospital and it was decided that surgical intervention was necessary, we were faced with the problem of no satisfactory homograft being available for replacement. A

reentry type of procedure to check the dissection, as advocated by DeBakey, was planned at the time the incision was made. When the size and condition of the aneurysm was determined, it was obvious that simple reentry of blood flow would not solve the problem. It would be just a matter of time until the left lateral wall of the aneurysm ruptured. During the procedure the entire hospital staff was alerted as to our urgent need for an aortic graft. It so happened that a patient died on the medical service during the course of the operation. Immediate permission for the use of the aorta was obtained, and it was removed under aseptic conditions. Unfortunately, it was very sclerotic and would not serve adequately.

It was then decided to construct a prosthesis. A crimped nylon bifurcation graft was available, and the bifurcation was removed, leaving the aortic portion approximately 16 cm. long. The ends were treated with the cautery to prevent fraying, and holes were burned in the nylon at the measured points for the celiac axis, superior mesenteric, and both renal arteries, respectively. Because of the difficulty in suturing vessels to the nylon once it was in place, and because the vessels had been foreshortened by the growth of the aneurysm, 3 cm. stumps of frozen-dried iliac homografts were sutured to the nylon to make the prosthesis complete with branches before it was sutured in place. This made suture to the normal vessels technically much less difficult and surer of success. It had the disadvantage of adding four more suture lines to the graft, thus increasing the chance for clotting to occur. A total of 10 vessel anastomoses were present at the completion of the graft.

The insertion of a by-pass around the aneurysm, as advocated by DeBakey,[1] is believed to be a wise approach and was attempted in this case. The proximal anastomosis was not properly made, however; and by the time the graft was inserted, the blood flow through the by-pass was inadequate. This necessitated reestablishment of aortic flow before any renal blood flow could be accomplished and added about 20 more minutes of ischemia to the abdominal viscera than should have been necessary. In retrospect, it would have been wiser to reanastomose the by-pass and make sure it was working properly.

Adequate exposure is mandatory in surgery of any type and can be obtained in the midabdominal aorta only through a combined thoracoabdominal incision. It is also necessary to incise the left peritoneal reflection of the colon and approach the aneurysm from the side. Dissection of the colon, pancreas, and stomach from the vessels and packing them to the right side of the abdomen provides good exposure.

Physiologic Considerations

The insult to the kidneys from prolonged ischemia is reflected in Figure 2. A discussion of this problem is of interest, since little is known of the time that human abdominal viscera can withstand ischemia and survive. Anuria of moderate severity developed in this patient, and it required 7 days before the urine volume began to rise and approximately 20 days before the blood urea nitrogen returned toward normal.

Van Slyke and associates[6] have shown that the dog kidney may recover entirely after complete ischemia of two hours' duration. It required at least 30 days for normal renal function to return. Koletsky[4,5] has studied this problem extensively in the rat and has found that normal function returns readily after two hours of clamping of the renal artery. Recovery was limited in longer periods of ischemia, and irreversible damage occurred with four hours of ischemia.

The first thoracoabdominal aneurysm resected by DeBakey had bilateral renal artery occlusion for 105 minutes.[1] The case presented here had occlusion of the left renal artery for 53 minutes and of the right renal artery for 70 minutes. It appears that good recovery may occur after renal artery occlusion of at least one hour, and perhaps upward of two hours, a period comparable to that in experimental animals.

The effects of ischemia on the bowel are more difficult to evaluate. This patient had no blood flow through the major vascular supply to his intestinal tract for a period of 1 hour and 40 minutes, and, although no clotting could be grossly demonstrated and the bowel "pinked up" normally upon reestablishment of the circulation, certainly profound depression occurred in the postoperative period. Adequate bowel sounds were not heard for seven days following the operation. However, distention of the bowel and fluid loss was never a problem. An inlying nasogastric tube for gastic decompression was used until bowel sounds were heard. Once bowel function returned, it appeared to be perfectly normal, and there was no evidence of gastrointestinal hemorrhage or mucosal slough in the stools.

The problem of possible clot formation in the intestinal vasculature during occlusion of the superior mesenteric artery suggested the use of an anticoagulant, such as heparin, to be given at the time of occlusion. It is probable that the heparin, if injected into the superior mesenteric artery at the time of occlusion, would, once circulation was reestablished, create problems at the graft site or in the operative wound. The heparin effect could be blocked by protamine at the time circulation was reestablished, but we did not believe that anticoagulation was a necessary risk.

The effects of ischemia on the liver, approximately of two hours' duration, are also difficult to evaluate. Liver function tests revealed a cephalin flocculation test of 2+ in the immediate postoperative period. This is in keeping with the studies of Child et al.[8] on total hepatic artery ligation in the monkey, in which the cephalin flocculation test rose to 1+ or 2+, this increase being the only abnormal finding. Once reestablishment of aortic blood flow had been carried out in this patient, some oxygenation of the liver probably occurred from portal blood flow. Since the inferior mesenteric artery was intact, immediate restoration of blood flow through the aorta would create circulation through part of the bowel and thus improve portal blood flow. Thus, it cannot be concluded that total hepatic ischemia occurred for the full two hours of the occlusion of the celiac artery.

Summary

The case of a 41-year-old man with a large thoracoabdominal aneurysm which had begun to dissect is reported. Because of the precarious situation of this patient at the time of admission to the hospital, it was decided to explore the abdomen and attempt to reenter the dissecting portion of the aneurysm. On exploration, however, a large aneurysm was found involving the thoracoabdominal aorta, including the celiac, superior mesenteric, and both renal arteries. Because the aneurysm was both dissecting and penetrating the wall of the aorta, it was believed that the only way to save the patient's life was to resect the aneurysm. A prosthesis was manufactured from a woven nylon aortic graft, onto which were sutured stubs of frozen-dried iliac arteries to provide the superior mesenteric, celiac, and both renal arteries. The aneurysm was then resected, and the graft was inserted and the blood flow reestablished. A discussion of the technical difficulties involved in the procedure and of the physiologic alterations in the patient's postoperative course is presented.

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Preoperative and Postoperative Care of Congenital Pyloric Stenosis

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Once the diagnosis of congenital hypertrophy pyloric stenosis has been definitely established by a history of projectile vomiting with palpation of the pyloric tumor by an experienced clinician, or if no tumor is palpated but an upper gastrointestinal series demonstrates the typical string sign and an elongated pyloric canal with gastric retention of barium, the main consideration is the preparation of the infant for the Fredet-Ramstedt operation. All these patients demonstrate a varying degree of dehydration, weight loss, and metabolic alkalosis, which need correction prior to surgical intervention. Only when an adequate formula can be taken by mouth and absorbed will the infant be able to replace the protoplasm he has lost and begin to grow.

The aim of preoperative treatment is to restore as quickly, but as safely, as possible most of the accumulated electrolyte and water losses; to ensure sufficient hemoglobin and protein content of the blood; to recognize and treat any complications, such as infection, and to operate upon an infant whose condition is adequate for surgery, which is usually between 24 and 48 hours after admission to the hospital.

The infant with pyloric stenosis has been vomiting an acid gastric juice plus his caloric intake. He may early be able to retain sufficiently to maintain his water and electrolyte balance but may only show no growth or loss of weight because of insufficient caloric balance. However, as the vomiting persists and compensating mechanisms are overcome, he develops an increasing deficit of sodium, potassium, chloride, and water. Since the gastric juice contains more chloride than sodium, the infant develops an increasing hypochloremic metabolic alkalosis, as demonstrated by an increasing blood bicarbonate content and a decreasing serum chloride. The alkalosis is often partially compensated by hypopnea or intermittent apnea, which will be seen only if looked for. The loss of potassium in the gastric juice plus the loss of potassium from the cells, due to the alkalosis, as shown by Darrow,[1] Danowski,[2] and their associates leads to significant potassium deficits. Mild deficits are asymptomatic, but infants with large deficits show a poor clinical condition and only partial response to parenteral saline and ammonium chloride. These large deficits are confirmed by a low serum potassium (after initial hydration) or by electrocardiographic changes. It is important to recognize that laboratory studies serve only as estimates of disturbed chemical changes, and their interpretation must be evaluated in the light of good clinical judgment.

With increasing alkalosis and dehydration from an accumulating deficit of water and sodium, up to 10% or more of the body weight may be lost. Decreased plasma volume causes poor circulation and impaired renal function, and finally shock may supervene.

Finally, with the alkalosis, decreased blood-ionized calcium may lead to tetany, usually manifested by convulsions, which throws an additional stress on an infant already in poor condition.

Parenteral fluid therapy varies with the severity of the deficits cited above. We have arbitrarily classified our cases as mild,

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moderate, and severe. In mild cases the infant often is not dehydrated, or only mildly so, and has no or slight metabolic alkalosis, with a carbon dioxide content of 32 mEq. per liter or less. In moderate cases the patients are usually dehydrated and have a carbon dioxide content of 33 to 42 mEq. per liter. In severe cases the carbon dioxide content is greater than 42 mEq. per liter or there is severe malnutrition or symptomatic hypopotassemia or tetanic convulsions. It is in the latter group that one's clinical judgment is put to the test. Mild and moderate cases are easily handled by a variety of regimes, and as long as reasonable amounts of parenteral fluids are given and renal function is not impaired, the infant will do well.

The severe cases demand the utmost attention and are a challenge to one's best clinical judgment. Occasionally one is confronted with tetanic convulsions. The infant is in danger of his life until they are stopped. A rebreathing bag or inhalation of 5% to 10% carbon dioxide in oxygen by mask with the slow intravenous administration of diluted calcium gluconate or calcium chloride will stop the convulsions.

The preoperative preparation as used in our institutions is as follows: If an infant is not dehydrated and is retaining some feedings, stock formula for premature infants may be fed every four hours until four hours before operation. In regard to preoperative fluid therapy, if an infant is retaining most of his fluids and the chemical studies are normal, and if he is hydrated and is in good general condition, only intravenous dextrose solution is given before operation. If blood chemical studies are abnormal, intravenous fluids only are given, and we give 5% to 10% dextrose solution in one-third strength saline (0.3%) intravenously plus added M/6 ammonium chloride subcutaneously. The latter is calculated as 2 cc. per pound of body weight of M/6 NH_4Cl for each milliequivalent the carbon dioxide content is above 30 mEq. per liter. This solution may be repeated, depending upon subsequent chemical studies. After hydration 10-20 mEq. per liter of potassium chloride is added to all intravenous and subcutaneous fluids. If there is evidence after hydration of hypopotassemia by serum potassium determination or electrocardiographic studies, 40 mEq. per liter of potassium chloride is added to the fluids. Total fluids given are 60-90 cc. per pound per day, plus amounts equal to 5% to 10% of body weight to replace dehydration loss when present. If the plasma protein is low, 10 cc. per pound of plasma is substituted for saline. If the hemoglobin is low, 5 to 10 cc. per pound of blood is substituted for saline.

A small Levin tube is passed into the stomach prior to the operation so that the stomach is entirely empty of fluid, gas, or barium sulfate. This is highly important in order to prevent aspiration during anesthesia. The Levin tube is left in place during the operation and withdrawn as soon as the infant has reacted from the anesthesia.

The postoperative therapy is mainly concerned with intravenous fluids, and these fluids are given to maintain an average fluid intake of 60 cc. per pound per day when the oral fluid intake is insufficient. This fluid is given as 5% to 10% dextrose in one-sixth-strength saline. The oral feedings are started four hours after surgery if the infant is alert and has a good sucking reflex. Ten per cent sugar water is alternated with one-half-strength evaporated milk formula every two hours, starting with 10 cc. and increasing 10 cc. every four hours until 2 oz. (60 cc.) is taken and retained at one feeding; and then the evaporated milk formula is increased to three-fourths strength and later to full strength. At the end of 32 hours the infant should be taking 2 oz. of formula. If vomiting occurs, there should be no increase, but formula should revert to the amount last given and retained. If vomiting continues, one of the following measures may be tried: small doses of phenobarbital, or chlorpromazine (Thorazine), or thickened feedings. If

there is much gastric mucus, we have found that washing the stomach out with warm saline usually will stop the vomiting. Vomiting in the postoperative period is not of serious moment and is not a major problem, provided all the pyloric fibers have been completely severed at time of operation.

During the past 17 years, the preoperative period of preparation of these patients has shown a progressive decrease from 3.5 days, from 1940 to 1945, to 1.8 days, during the past six years. The postoperative hospital stay has also been decreased from 13.5 days, in 1940 to 1945, to 5.4 days, during the past six years. Approximately 65% of our patients now enter the hospital in a minimal degree of metabolic alkalosis, 15% with a moderate degree of alkalosis, and 10% with a severe degree of metabolic alkalosis or severe malnutrition.

In our experience with the Fredet-Ramstedt operation for congenital pyloric stenosis at our institutions during the past 17 years (1940-1956, inclusive), 742 infants have been operated upon. There have been six deaths following operation, or a mortality of 0.8%. Three deaths were due to intercurrent diarrhea; one, to an overlooked minute duodenal perforation with subsequent peritonitis; one, to a possible adrenal insufficiency on the second postoperative day, and one, to extensive liver necrosis, of unknown etiology, on the sixth postoperative day.

The last two deaths occurred in infants admitted in severe states of malnutrition. Correction of their fluid and electrolyte disturbance was attained before operation. We believe their severe and prolonged state of malnutrition was the main factor in their failure to survive.

Summary and Conclusions

With earlier diagnosis of congenital pyloric stenosis, infants require a shorter preoperative stay.

A more effective and rapid and safer correction of metabolic alkalosis has shortened the preoperative period.

Infants with minimal, moderate, and severe degrees of metabolic alkalosis are seen, but infants in the last group are becoming less frequent.

Better preoperative and postoperative fluid-balance control has been an important factor in maintaining a low mortality rate, while reducing the hospital stay.

The operations have been performed on 742 infants over a 17-year period, with a mortality rate of 0.8%.

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Intestinal Obstruction of Congenital Origin

A Study of Diagnosis and Management in One Hundred Sixty-Three Cases

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Physicians concerned with the care of children today are aware of the increasing importance of developmental malformations as a cause of death and disability in early life. Fifteen per cent of these malformations involve the gastrointestinal tract,[1] and the majority of these lesions produce some form of intestinal obstruction, which, if complete, can cause or contribute to the death of the infant in the neonatal period; if incomplete or of recurring nature, can lead to prolonged disability throughout infancy, childhood, and, occasionally, adult life.

Intestinal obstruction of congenital origin may be due to (1) intrinsic obstruction (atresia and stenosis), (2) neuromuscular defects (primary intestinal aganglionosis), (3) anomalies of rotation and fixation (malrotation, volvulus, internal hernia, congenital bands), (4) intraluminal obstruction (meconium ileus or meconium plugs), (5) extrinsic lesions (annular pancreas, duplication, neoplasm), (6) and meconium peritonitis (Charts 1 and 2). Not infrequently more than one of these lesions may exist in a single patient; yet in most instances a correct preoperative diagnosis can be established if a thoughtful study of each case is made.

The outlook today for these patients is immeasurably improved over that of a decade ago, since personnel and institutions trained and qualified to meet the exacting requirements of the very young are now more widely available. Further improvements in infant salvage can probably best be achieved by emphasizing the importance of early diagnosis, based upon a careful evaluation of the history and physical examination, proper radiographic studies, and certain laboratory data. The importance of

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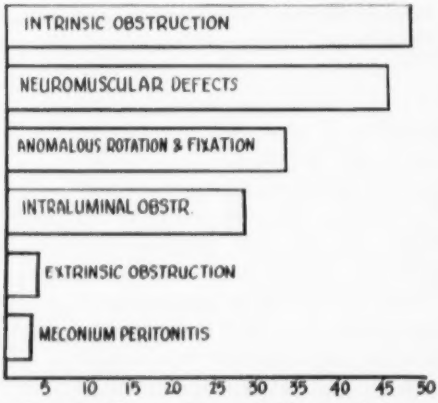


Chart 1.—Causes of congenital intestinal obstruction in 163 cases in the Columbus Children's Hospital series.

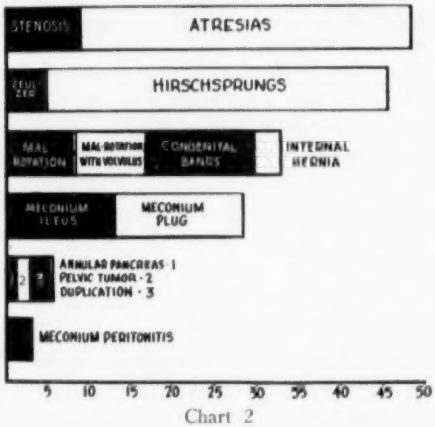


Chart 2

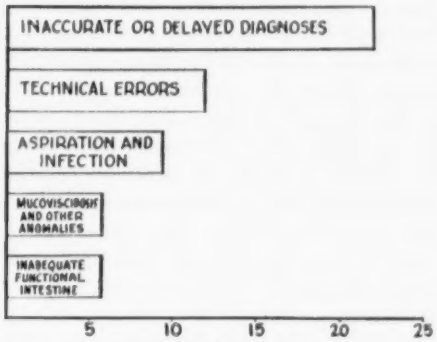


Chart 3.—Causes of 56 deaths due to congenital intestinal obstruction (49 autopsies).

these particular details of care have been clearly emphasized for us by a recent survey of a series of 163 consecutive cases of intestinal obstruction of congenital origin in infants who were admitted to the Children's Hospital, Columbus, during the 12-year period 1944-1956. Unfortunately, 56 of these infants died, 43 of what may be considered in retrospect avoidable causes.

The major contributing factor in the deaths of 22 infants was delayed or inaccurate diagnosis; in 12 instances, errors in judgment or surgical technique at the operating table or during the precarious pre- and postoperative period led to the fatality, and in 9 instances sepsis appeared to play the major role. The remainder of the deaths can perhaps best be classified as unavoidable, since they were due to the pulmonary and nutritional complications of fibrocystic disease, to associated major congenital anomalies incompatible with life, and, in a few instances, to inadequacy of the remaining gastrointestinal tract or to such extensive in utero peritonitis that salvage appeared impossible Chart 3).

In presenting this paper, therefore, it seems appropriate to emphasize the problems which are related to the early recognition of intestinal obstruction in the infant and to the methods of evaluation peculiar to this age group, and to consider briefly surgical management of each specific entity. No effort will be made to discuss in detail the established surgical techniques, which are so well documented in standard texts and current publications on this broad subject.[2-4]

Diagnosis of Intestinal Obstruction

The first step in the successful management of any pathologic entity is the establishment of an accurate diagnosis. Since over half of the avoidable deaths which occurred in this series of patients were the result of incomplete or late diagnosis, we have carefully reviewed the clinical course of this disease as it presents itself in the newly born.

The four cardinal manifestations of congenital intestinal obstruction are abdominal distention, bilious vomiting, abnormality of meconium evacuation, and maternal polyhydramnios (Table).

Polyhydramnios.—Obstetric and newborn nursery personnel are, in general, not sufficiently alert to the fact that polyhydramnios in a mother is often the first evidence of alimentary-tract obstruction in the fetus. When the volume of amniotic fluid exceeds 2000 cc.[5] and there are no obvious abnormalities in the infant, obstructive disease should be immediately suspected. Ingalls and Prindle,[6] in a recent review, have reported this condition to be present in 25% of the cases of atresia of the esophagus. Similar findings were noted in 18% of our own series of infants with esophageal atresia and in 45% of the patients in this series who had duodenal or high jejunal obstruction.[7] By contrast, polyhydramnios is rarely

Summary of Cardinal Manifestations of Congenital Intestinal Obstruction Observed in 163 Cases

Polyhydramnios	Vomiting		Distention		Stools	
17 cases, 45% with duodenal or proximal jejunal obstruction	None	39	No	34	Normal	16
	Present	124	Present	129	None	56
	Bile	83	Upper	22	Scant	70
	Fecal	5	Generalized	107	Diarrhea	15
	Blood	2			Blood	6

associated with obstructive lesions of the lower reaches of the small or large intestine, presumably because in such cases there is sufficient absorptive surface available to assist in the transfer of the amniotic fluid to the mother's circulation.

Abdominal Distention.—Abdominal distention was present at birth or developed during the course of the disease in 129 of the infants included in this series. The nature of the distention varied. In 22 patients with high obstruction it was localized to the upper abdomen, whereas in 107 infants with ileal or colonic lesions it was more generalized. When abdominal distention was associated with prominence of the veins over the abdominal wall, obstruction was presumed to be present in utero.

Vomiting of Bile.—Although the spitting up of small amounts of mucus and gastric fluid in the first few days of life is commonplace, the regurgitation or vomiting of bile-stained material is rarely seen in the absence of mechanical obstruction. In this series 124 of the infants vomited, and the vomitus was bile-stained in 83 instances. Bilious vomiting occurs earlier and is more profuse in infants with high obstruction than in those with lesions in the lower small bowel or the colon.

Abnormality of Stools.—The fetal colon accumulates the residue of the in utero alimentation and at the time of birth contains 60 to 200 gm. of thick, viscid meconium. Failure to initiate the evacuation of meconium within the first 24 hours after birth is distinctly unusual, and the last of the meconium will ordinarily be passed within 72 hours.[8] Occasionally, infants with incomplete or high obstruction may pass small amounts of light-colored, grayish, or gray-green meconium-like material, which should not be confused in the nurses' notes or in the physician's mind with a truly normal evacuation.

In certain infants, particularly the premature ones, rectal stimulation by thermometer or by a careful digital examination may be helpful in initiating meconium evacuation. However, if this simple and safe method fails, a more complete diagnostic work-up is necessary. Repeated colonic irrigations prior to the initial radiographic studies should be avoided, since the injection of air and fluid in the colon may confuse the diagnosis, as well as afford an opportunity for perforation of the small, thin-walled bowel.

In this series, 56 of the infants had no stools whatever; 70 passed small amounts of material by rectum or were severely constipated or obstipated by their obstruction during the first few weeks and months of life; 15 had diarrhea; 6 passed gross blood by rectum, and 16 had what was described as normal bowel habit.

It is apparent that these manifestations are readily recognizable and that the medical personnel responsible for the delivery and care of the young infant should be alert to these evidences of obstructive disease. Any infant with any one of the cardinal symptoms or signs of obstruction of the alimentary tract deserves a planned regimen designed to establish or exclude the diagnosis.

History and Physical Examination

The history, family history, and physical examination are of limited value in determining the exact nature of the obstructive lesion in the newly born. Unfortunately, the details of the pregnancy and immediate postpartum period are often too incomplete for careful evaluation. In older infants and children the nursery notes or the mother's observations may be of inestimable value in determining whether the child has chronic or acute obstructive disease. It is particularly important to be aware of the sudden onset of symptoms in an infant who has previously been well and suffers a cataclysmic change characteristic of volvulus or internal hernia, which so often results in gangrenous obstruction.

The family history is helpful in the differential diagnosis of meconium ileus and primary aganglionic megacolon. Intestinal

obstruction due to meconium ileus is the earliest clinical manifestation of fibrocystic disease of the pancreas and occurs in 10% to 20% of the cases. As a recessive genetic lesion this disease would be expected to occur in 25% of any given gene-mating. The familial tendency to aganglionic disease is less clearly understood but has been reported in siblings. We have recently cared for three brothers with typical clinical and pathologic evidence of this common congenital malady. There have been three other patients in our series who had siblings who died in infancy of an undiagnosed "intestinal disorder." The majority of lesions producing intestinal obstruction of congenital origin appear to be unrelated to heredity and presumably result from acquired errors in embryonic development.

The physical examination should include an appraisal of the patient's general condition, a search for additional major malformations, and a careful evaluation of the abdomen. Of particular importance is the nature of the distention, the size and the position of the gas- and fluid-filled loops, which will often suggest the level and type of the obstruction. Occasionally, meconium-distended loops of bowel or irregular pellets of abnormal meconium debris may be felt in the ileocecal area in obstruction due to meconium ileus.

Peristalsis is normally visible, as well as audible, in these thin-walled abdomens. Hence, unless bowel contours and the peristaltic activity are extremely prominent, little significance can be attributed to it. Absence of peristalsis is uncommon and usually is associated with edema of the abdominal wall, gangrenous obstruction, perforation, and peritonitis.

The examination of the anus and rectal canal should be accomplished with care. If the anal opening is perforate and in normal position (not anterior to the anal sphincter), the anus is probably normal. When the finger is slowly introduced through this sphincter, one may occasionally completely relieve an obstruction at this level in patients suffering from the meconium-plug syndrome and, to a temporary and less marked extent, in true aganglionic Hirschsprung's disease when the lower rectal segment alone is involved. However, if the obstruction is higher up, the examiner will get the impression that the rectal canal is stenotic or strictured, since it has never been distended by the fecal stream.

No physical examination of a newborn infant who is suspected of having intestinal obstruction is complete until a stiff No. 8 or No. 10 F. urethral catheter has been passed into the stomach to prove patency of the esophagus and to determine the character of the gastric aspirant.

Radiographic Examination

The radiographic examination furnishes the most accurate means of evaluating an infant suspected of having intestinal obstruction, provided the clinician is aware of the bizarre anatomical changes which may result from lesions that occur in the newborn. The human infant's gastrointestinal tract is anatomically complete, enzymatically active, and at least partially functional by the end of the first trimester of in utero existence. During the ensuing months the fetus continuously ingests and assimilates the amniotic fluid which surrounds it.[9]

Obstruction to the upper and, to a less extent, the lower gastrointestinal tract will seriously affect the normal pattern of growth and development. Extreme degrees of dilatation and hypertrophy of the bowel will develop proximal to the site of the obstruction, while the distal bowel, with has never been used, will remain tiny and thin-walled (Fig. 1). Not infrequently necrosis and perforation result in meconium peritonitis and the development of calcification and massive vascular adhesions throughout the peritoneal cavity. These changes which occur during intrauterine life lead to rather unintelligible clinical and radiographic findings, which are quite unlike those conventionally seen in acquired obstructive disease in later life.

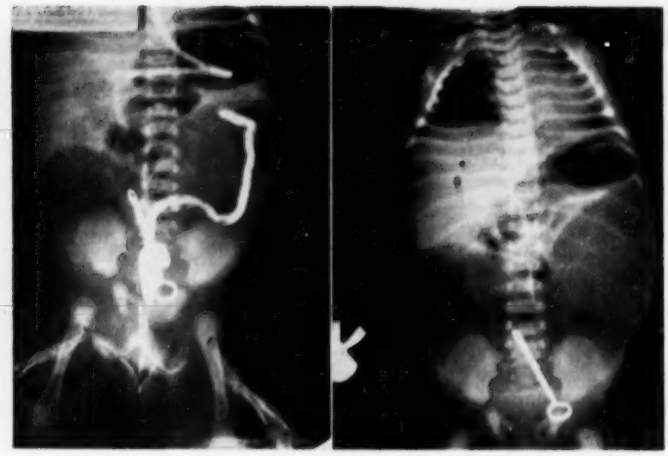


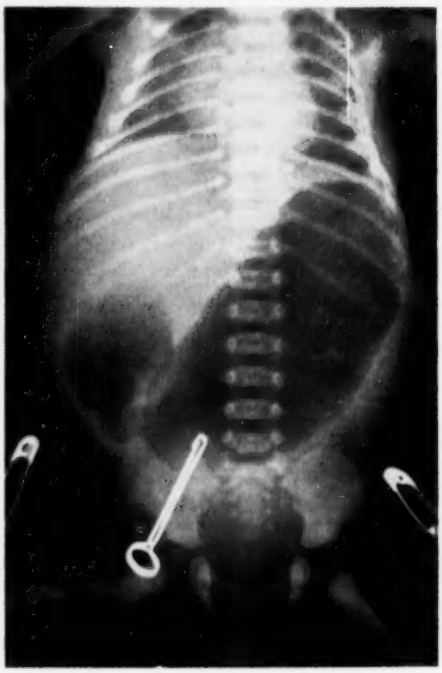
Fig. 1.—Plain-film and barium-enema study of a newborn infant with atresia of the ileum demonstrating, left, the huge dilatation of the small bowel proximal to the obstruction, and, right, the tiny, unused colon distal to the obstruction.

Fortunately, nature provides the radiologist with an excellent contrast medium in the form of swallowed air. Normally the stomach fills with the first breath; the duodenum, within a few minutes, and the air column will reach the cecum in a normal infant within three to eight hours.[10] The initial radiographic examination should consist of a flat and an upright film of the abdomen and chest. In many instances these two simple exposures permit a complete assessment of the problem. Occasionally films with the patient in the lateral decubitus or upside-down position are helpful. High-grade, high-intestinal obstruction, such as stenosis or atresia of the duodenum, usually produces a "double-bubble" or single, huge U-shaped fluid- and air-filled loops with an absence or paucity of gas below this point (Fig. 2).

Free air in the peritoneal cavity is indicative of perforation. An excessive amount of intraperitoneal fluid is usually associated with acute gangrenous obstruction, such as internal hernia or midintestinal volvulus. Intraperitoneal calcification is pathognomonic of meconium peritonitis; occasionally tubular calcifications are seen, which are believed to be evidence of previous ischemia or necrosis of the bowel wall.

In patients with meconium ileus the intestinal loops vary in size, and in a few cases there will be seen mottled or foamy masses of meconium, as described by Neuhauser.[11] The mottling is due either to air bubbles or to inspissated fatty material, which abounds in this substance in the absence of lipolytic enzymes. In an even larger proportion of the cases, Zimmer[12] and, more recently,

Fig. 2.—Plain film of an infant with duodenal atresia demonstrating the huge dilatation of the stomach and the duodenum (double bubble).



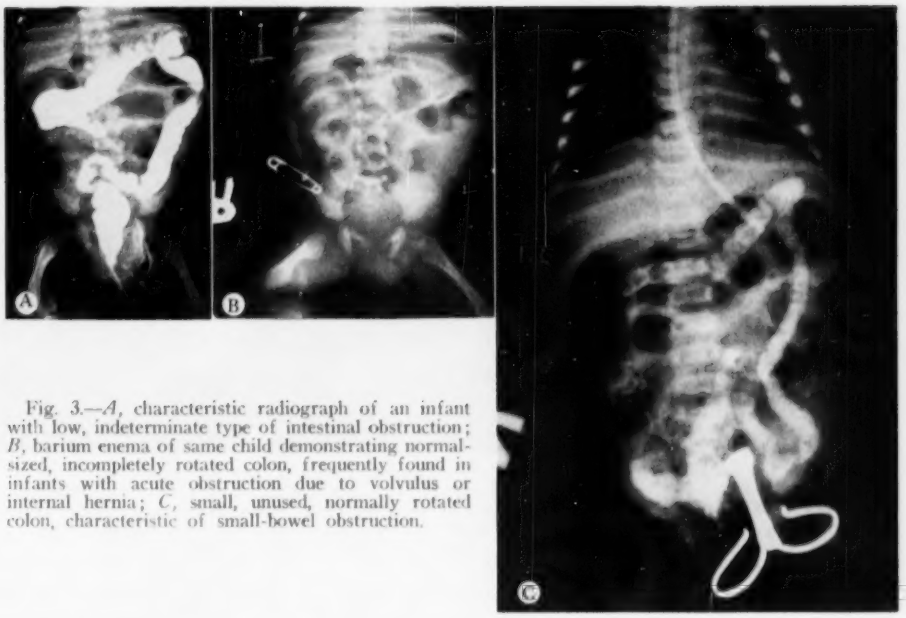


Fig. 3.—*A*, characteristic radiograph of an infant with low, indeterminate type of intestinal obstruction; *B*, barium enema of same child demonstrating normal-sized, incompletely rotated colon, frequently found in infants with acute obstruction due to volvulus or internal hernia; *C*, small, unused, normally rotated colon, characteristic of small-bowel obstruction.

White [13] have called attention to the characteristic lack of fluid levels in this condition.

In the event that the plain radiographs are not diagnostic, the barium enema is next most useful in examination of infants. We have been discouraged and disillusioned in our efforts to differentiate between lower-small-bowel and large-bowel obstructions in young children with plain film studies alone. For this reason, we recommend the performance of a barium-saline enema on every infant with a low, indeterminate type of intestinal obstruction.

The barium enema will differentiate large- from small-bowel obstruction, will indicate the type of colonic obstruction that may exist, and will demonstrate whether or not intestinal rotation is complete. In the presence of small-bowel obstruction, the colon will be unused and micro-sized (Fig. 3). If the lesion involves the large bowel, the point of obstruction will be demonstrated by the barium column. In an infant with the meconium-plug syndrome, the colon will be narrow up to the point of the plug (usually in the region of the splenic flexure). Expulsion of the plug may follow the barium enema, and subsequent studies will demonstrate progressive enlargement of the previously narrowed colonic segment. In true Hirschsprung's disease the distal colon may be small initially, but there will be a progressive caliber change between the narrowed aganglionic rectosigmoid and the proximal normal bowel. In the rare instances of diffuse aganglionic disease, the barium will be segmented and retained in the colon for several days (Fig. 4). When rotation is incomplete, the infant may have midintestinal volvulus, in addition to such commonly associated defects as paraduodenal or intraperitoneal bands and transmesenteric hernia.

Management of Specific Lesions

The preoperative management of infants and young children with intestinal obstruction requires the utmost diligence and vigilance. Prior to surgical intervention, the blood volume defects should be corrected and any major aberrations of water and electrolyte balance remedied. Overhydration, aspiration pneumonia, and infection are the

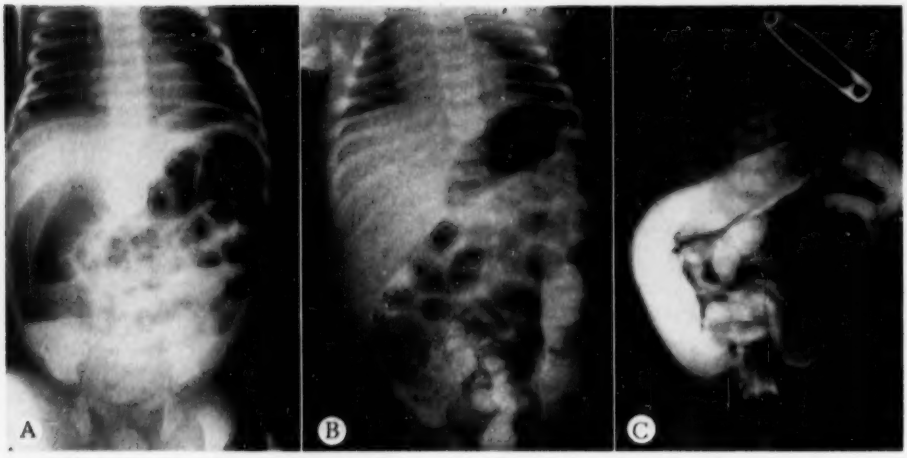


Fig. 4.—*A*, upright radiograph of 2-day-old infant with low, indeterminate type of intestinal obstruction; *B*, barium-enema study in the same patient with early radiographic evidence of true aganglionic megacolon, which was proved by rectal biopsy and treated by temporary colostomy; *C*, barium-enema study in 3-day-old infant with the meconium-plug syndrome (note plug at splenic flexure). Permanent relief by barium enema.

major problems to be combated in these patients. Frequent careful weight determinations, the proper choice of antibiotic agents, and continuous supervision by trained nursing personnel in clean surgical units are the best assurance against unnecessary deaths.

Causes of Congenital Intestinal Obstruction

Intrinsic Obstruction.—Atresia or stenosis of the intestine was the most commonly encountered lesion in this series and occurred in 48 cases. There were nine cases of stenosis, eight in the duodenojejunal area. By contrast, the 39 cases of atresia were more widely distributed—17 duodenal, 10 jejunal, 10 ileal, and 2 in the colon. These lesions varied from thin mucosal webs to complete separation of segments of the bowel, and were often multiple. Mongolism occurred in eight cases of duodenal stenosis or atresia.

Various surgical procedures were used to establish intestinal continuity. Isoperistaltic side-to-side anastomosis, using two layers of interrupted fine silk sutures, was preferred in all duodenal lesions. For lesions below the ligament of Treitz we now prefer an oblique end-to-end enteroenterostomy, in an effort to avoid the late complication of "blind-loop syndrome" which may occur distal to a side-to-side anastomosis. As emphasized by Bill and Pope,[13] it is our usual policy to open all widely dilated duodenums to avoid missing an intrinsic web.

Lesions of the lower jejunum and ileum present the greatest technical problems. We have become discouraged with the Mikulicz type of three-stage procedure. We are now exploring the desirability of carrying out more extensive resections of the proximal abnormally dilated or the distal micro-sized intestine. Such a maneuver permits the surgeon to use essentially normal bowel to restore intestinal continuity by a proximal end-to-end enteroanastomosis or a distal end-to-side ileocecostomy. This approach permits adequate decompression by nasogastric intubation or enterotomy drainage through the appendicocele stump during the immediate postoperative period.

That such extensive resections may be warranted is supported by experimental studies performed in our laboratory in growing puppies which showed that the resection of as much as 75% of the small

bowel was well tolerated.[15] In this series there were five infants who underwent the resection of 21 to 160 cm. of small bowel (normal length of small intestine at birth, 300 cm.) and recovered.

The over-all mortality in this group of infants with all types of intrinsic obstruction was 46%.

Neuromuscular Defects.—Forty-six infants and children were considered to have primary aganglionic disease of the colon or small bowel or both. In each case the symptoms dated from birth, although 22 of these children did not come to our institution for care until they were 6 months of age or older. In 44 cases the aganglionosis was confined to the rectosigmoid area (true Hirschsprung's disease). In one instance the entire colon was involved, and in one case the entire alimentary tract was deficient in ganglion cells.

Infants with Hirschsprung's disease may experience considerable difficulty in initiating the evacuation of the meconium and suffer from recurring episodes of distention, constipation, and obstipation. Medical management, including dietary changes, laxatives, parasympathomimetic drugs, and repeated rectal irrigations, has been widely used in the initial treatment of these children. The complications of such treatment include perforation of the colon (three cases in this series), water intoxication, growth failure, parental anxiety, and a poorly understood susceptibility of such infants to severe enterocolitis, which does not respond to the usual modes of management of diarrheal disease. For these reasons, we now recommend that symptomatic infants with primary megacolon have a temporary loop colostomy performed in the sigmoid area as soon as the diagnosis is established by barium enema or rectal-wall biopsy. When the child is 18-24 months of age, a primary Swenson[16] type pull-through procedure is then done to restore normal colonic function.

There were 10 deaths in the patients with aganglionic disease. Two patients had extensive disease involving the entire colon or alimentary tract. Four died of progressive obstruction or perforation of the colon while on medical therapy. Three infants died prior to 1950, following an exploratory laparotomy for "intestinal obstruction." At operation no definite obstruction could be demonstrated. A diagnosis of volvulus of the sigmoid or constricting bands was made and the abdomen was closed without relieving the obstruction. One infant, aged 6 months, succumbed in 1949 to pelvic peritonitis following a primary pull-through procedure. The remaining 25 patients are still living following a definitive type of surgical procedure or are in various stages of treatment.

Anomalies of Rotation and Fixation.—The surgical management of the lesions resulting from anomalous rotation or fixation has presented a few problems. If the diagnosis is made sufficiently early and the intestine is viable, the expected mortality should be very low. The two major technical problems pertain to the need for recognizing associated intrinsic obstruction and the management of infants with necrosis of a large segment of the midintestine. In three cases intrinsic duodenal obstructions were missed by the surgeon at the time the Ladd procedure was performed. In each of these cases a review of the x-rays indicated that the proximal duodenum was more widely dilated than is usually found with malrotation and paraduodenal bands alone. Since that time we have opened all the widely dilated duodena, even though definite bands associated with malrotation may be present (Fig. 5). In cases with necrosis of a large segment of the small intestine, a resection should be carried out. Three such cases of volvulus, in which the patients have survived the removal of all but a few inches of proximal jejunum and distal ileus, have recently been reported by Pilling[17] and by Kasmersky.[18] Obviously, such patients require prolonged careful hospital management while the adaptation of the remaining small bowel is taking place.

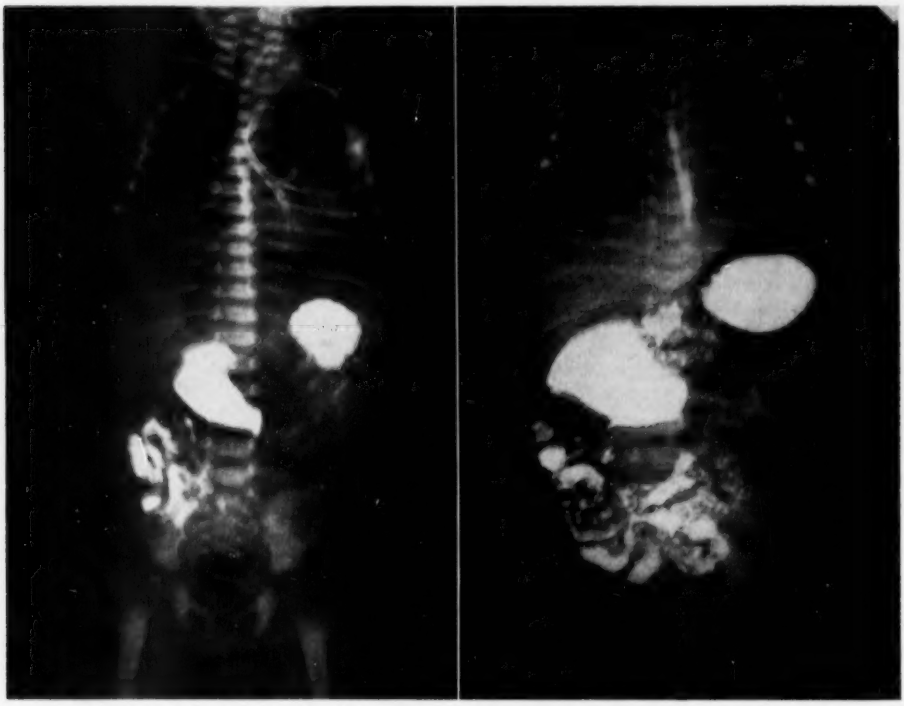


Fig. 5.—G. I. series of two infants previously demonstrated to have malrotation of the colon: left, moderately dilated duodenum, due to constrictive bands (relieved by Ladd's procedure alone); right, hugely dilated duodenum, suggesting that intrinsic stenosis is present, as well as paraduodenal bands. (Ladd's procedure and duodenojejunostomy were required to relieve obstruction.)

Of the 14 deaths in this group of patients, 11 resulted from a failure to suspect the diagnosis until the entire midintestine was noviable. In six cases the lesion was found at autopsy.

A review of the records of these unfortunate infants indicates that the majority were considered to have infectious diarrhea. Bile-stained vomiting was almost uniformly present early in the disease, but in the absence of distention and in the presence of diarrhea, insufficient attention was paid to this finding until too late. In six infants the serious nature of the alimentary-tract disturbance was not suspected until gross blood appeared in the stools; all of these infants died.

Intraluminal Obstruction.—In 28 newborn infants intestinal obstruction was produced by an obturating mass of viscid meconium. In 12 instances the meconium mass was localized to the mid- or terminal ileum, and in each case subsequent studies demonstrated other evidence of pancreatic fibrosis. These infants were variously treated by enterostomy and extrusion of the meconium, instillation of pancreatic enzyme, peroxide irrigations, and three-stage exteriorization resections, all with about equally good (or poor) results. By such means the obstruction could be relieved. Seven were discharged from the hospital. All have subsequently succumbed to or suffer from typical respiratory complications of their generalized disease.

By contrast, there were 14 cases in which the obstructive mass was confined to the transverse or left colon, as seen in the meconium-plug syndrome.[19] In each instance, although the initial gelatin-film-test

studies for tryptic activity indicated there was an absence of the enzyme gelatinase in the meconium, subsequent stools showed the presence of this enzyme. These 14 patients, who have been followed up to six years, have not shown any evidence of pulmonary or alimentary dysfunction commonly associated with pancreatic disease or any subsequent abnormality of colonic function, such as that seen with true Hirschsprung's disease.

Extrinsic Obstruction.—In four cases there was an extrinsic obstruction of the bowel other than that due to bands with malrotation. An annular pancreas was found in two cases; a duplication of the ileum and an angioma of the sigmoid colon, in one case each. In infancy the annular pancreas is frequently associated with an intrinsic obstruction of the duodenum as well, and in one case was successfully treated by duodenojejunostomy. No effort should be made to divide the abnormal pancreatic tissue. The infants with duplication of the terminal ileum and angioma of the sigmoid have survived excision of the local lesion.

Meconium Peritonitis.—Meconium peritonitis may frequently complicate other types of intestinal obstruction, particularly atresias and meconium ileus. However, we had three cases in which we could find no cause for the obstruction other than the massive adhesions which resulted from what appeared to be spontaneous perforation of the bowel in utero. Severe interference with intestinal motility was produced by the massive plastic and partially calcified peritoneal exudate present throughout the abdominal cavity. All infants in this series eventually succumbed to chronic malfunction of the intestinal tract, presumably due to persistent fibrinous peritonitis. Unless obstruction is manifest, we do not recommend surgical intervention in newborn infants with radiographic evidence of meconium peritonitis.

Summary

One hundred sixty-three consecutive cases of intestinal obstruction of congenital origin admitted to the Children's Hospital, Columbus, between 1944 and 1956 are reviewed. The causes of obstruction fall into seven major categories: intrinsic, extrinsic, and intraluminal; anomalies of rotation and fixation; neuromuscular defects, and meconium peritonitis.

There were 56 deaths in this group, 43 of which may be considered avoidable. Diagnostic inaccuracies accounted for 22 deaths, technical errors in surgery and management for 12, and infection for 9. The remaining deaths were from unavoidable causes, such as associated anomalies, complications secondary to mucoviscidosis, and inadequate intestine.

Early diagnosis is of paramount importance. One should suspect intestinal obstruction in any infant who vomits bile, is distended, has dilated abdominal-wall veins, fails to pass meconium, or is delivered of a mother with polyhydramnios.

X-rays are the most efficacious diagnostic aid. Flat and upright roentgenograms should be obtained on all patients suspected of having intestinal obstruction, and careful evaluation of gas shadows, mass shadows, fluid levels, and calcifications is diagnostic in the majority of the cases. In low or indeterminate obstruction the barium enema is necessary to differentiate large- from small-bowel obstructions and Hirschsprung's disease from meconium-plug syndrome.

The general management is aimed at (1) establishing fluid and electrolyte homeostasis and correcting hypovolemic states, (2) early definitive surgical intervention, with restoration of the anatomical gastrointestinal continuity without undue soiling, and (3) careful postoperative management. Experience has indicated that extensive resection of small bowel can be carried out without serious permanent growth impairment. The end-to-end or end-to-side enteroenterostomy is preferred for restoring continuity except in cases of duodenal obstruction or meconium ileus. Early temporary colostomy is recommended for symptomatic infants with primary aganglionic megacolon.

Attention to these details has resulted in a reduction of mortality from 52%, in 46 patients seen prior to 1951, to 27.4%, in 118 infants cared for from 1951 through 1956.

Dr. William Howard, Chief, Department of Radiology at the Children's Hospital, and Miss Brenda Olson, of the Departments of Medical Illustration and Photography of the Ohio State University College of Medicine, assisted with the roentgenologic aspects of this paper.

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Observations on Undescended Testes

I. Significance of the Empty Scrotum and Indications for Orchiopexy

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The confusion concerning the management of the undescended testis is not rivaled by many other lesions. When such disparity of opinion exists, it is safe to conclude that either the data available are hopelessly muddled or the observers are not all talking about the same thing.

The undescended testis should be a lesion which lends itself to thorough investigation from which definitive statements could be made concerning etiology, anatomy, and management. Instead, there are widely divergent views concerning causation; association with other lesions, such as hernia; medical versus surgical management; the indications for surgery; the type of operation needed; the age of election, and the results.

My associates and I have been interested in undescended testes for seven years and know that such a short period of observation cannot be definitive in all aspects of the problem. However, there are some observations we are now able to make on the basis of approximately 750 undescended testes seen at the Children's Hospital of Philadelphia, and several long-term studies are underway which will be reported subsequently.

It is the empty scrotum, unilateral or bilateral, which brings the youngster to the physician's attention. There are several possible explanations for an empty scrotum. The testis may be absent, atrophic, ectopic, or abdominal, in which instances the gonad will not be felt along the line of normal descent in the inguinal canal or in the scrotum. When the testis is not palpable, there is no way of ascertaining which of these possibilities exists except to explore the anatomical areas in question.

Occasionally in an obese youngster with an empty scrotum, an undescended testis lying low in the inguinal canal cannot be felt, even on repeated attempts, and yet turns up at operation as a true, undescended testis, not palpable because of overlying fat.

Misconceptions Concerning Testicular Descent

The crux of the discussion centering about the empty scrotum is in the gonad that can be palpated in the inguinal canal. Is this an undescended testis? The answer is "yes" only if on proper manipulation the testis cannot be pushed into the scrotum and brought to the very bottom of that elastic structure.

If the testis can be brought into the scrotum, even though when released it retracts immediately to midinguinal canal, it is not an undescended testis but, rather, is a physiologically retracted testis, and the patient stands in need of neither medical nor surgical care in this regard.

There are many widely held misconceptions concerning testicular descent. Among these are the following:

1. The testis begins its descent in utero and may not complete it until puberty.

2. Gonadotropic hormones bring this descent about naturally.

3. When a testis fails to descend naturally, the administration of gonadotropic hormones will hasten or ensure descent.

4. If a combination of time and hormones will not bring about the descent of the testis, surgical correction of the defect is advisable.

From the Surgical Clinic of the Children's Hospital of Philadelphia and the Harrison Department of Surgical Research, the University of Pennsylvania School of Medicine.

These misconceptions are based primarily upon the failure to differentiate the undescended testis from the retracted testis which is held high in the path of normal testicular descent by a hyperactive cremasteric reflex. Few realize that the anatomical configuration of the undescended testis, its relations to the tunica vaginalis testis, and its association with barrier between the inguinal canal and the scrotums are identical with the anatomical findings in human fetuses of 20-24 weeks' gestation.

In short, failure to recognize a true undescended testis as an embryologic failure has led to the confusion centering around this lesion. Undescended testis is a congenital defect just as certainly as is an imperforate anus, and there is no more reason to believe that time or medical management will correct this than there is to believe that an imperforate anus could be cared for without surgical intervention.

Differential Diagnosis of Retracted and Undescended Testis

The differential diagnosis of undescended testis and retracted testis is not difficult. That the two are readily confused is evident from the number of children referred to the Surgical Clinic of the Children's Hospital of Philadelphia with a diagnosis of undescended testis who on examination are found to have a normal physiologic situation, a retracted testis.

Given a child with an empty scrotum, examination should be carried out with the youngster on his back. Palpation of his scrotum should begin with gentle grasping of the entire hemiscrotum in the first three fingers of that hand of the examiner that corresponds to the side of the patient being examined (Fig. 1). If the testis is in the scrotum, it should be possible to bring it to the very bottom of the scrotum, even though that structure should become elongated in the process. The testis should not be forcibly squeezed during this palpation, but, rather, the fingers should close above the testis. If the scrotum is empty on the side being examined, the inguinal canal should be palpated, with the index and third finger of the opposite hand searching for the gonad (Fig. 2). When the testis is encountered, it should be pushed gently toward the scrotum, using either thumb and forefinger or the index and third finger (Fig. 3).

If the testis is merely retracted, and not undescended, the gonad will slip through the top of the scrotum, and it can then be picked up by the other hand and brought to the bottom of the scrotum (Fig. 4), as described in Figure 1.

Fig. 1.—Three fingers closing over the top of the testis and pulling it to the bottom of the scrotum.

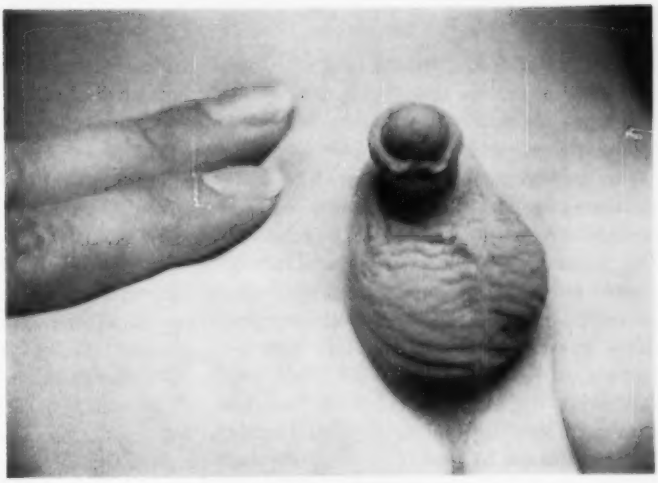

Fig. 2.—Pressure applied alternately by index and third fingers aids in finding the elusive testis.

Fig. 3.—Thumb and forefinger pushing testis, in snowplow fashion, into the scrotum.

Fig. 4.—The inguinal testis, having been delivered to the scrotum, is pulled down by the fingers of the other hand.

When repeated attempts fail to push the testis into the scrotum by this technique, it is safe to call the testis undescended, an embryologic failure.

For reasons which are not clear, such an undescended testis is usually arrested cephalad to the pubic tubercle. The relatively few instances in which the testis can be pushed into the upper scrotum but not pulled all the way to the bottom of that structure, tempt one to see how it will fare with the passage of time if left alone. We have watched a number of these testes and are convinced that they do not descend further. It is our custom to treat them as an inguinal undescended testis, to do a formal orchiopexy, and to repair the hernia if present.

The external appearance, as well as palpation, of the scrotum lends confirmatory evidence to the diagnosis of undescended testis. When a testis is simply retracted, it has already made its way embryonically into the scrotum and that structure has a cavity. When a testis is undescended, the embryonic processus vaginalis has never descended to produce a cavity in the scrotum, and this can usually be ascertained externally, but always proved by dissection.

Our experience indicates that an indirect inguinal hernia is found in 89% of cases of undescended testis. When one reads of reports, such as that of McCutcheon,[1] stating that hernias were found in only 4 of 204 cases of cryptorchism, one wonders, in view of all the evidence to the contrary,[2-4] whether this series was not predominantly one of retracted testes, and not of undescended testes.

The scrotum which has never been occupied by a testis not only is empty but frequently has a flattened appearance laterally when compared with the other side. Rubbing the scrotum between the fingers fails to reveal the usual slippery sensation elicited by the same maneuver in a scrotum where the testis has normally descended. Dissection of the two sides of the scrotum in a patient with one undescended testis reveals that on the normal side there is a cavity lined with a glistening mesothelial layer, while on the side of the undescended testis there is no such membrane and no cavity.

This very logically leads one to a consideration of how the testis gets into the scrotum in the first place. Hutchinson[5] has reported his observations on normal descent based on a study of approximately 200 stillborn infants, ranging in size from 16 to 40 cm. in crown-rump length, only a brief summary of which will be given here. The testis descends retroperitoneally from the posterior abdominal wall to the pelvic brim, where it follows the processus vaginalis through the inguinal canal and into the scrotum. The processus vaginalis is an evagination of the peritoneum through the internal and external rings, and it always precedes, to a slight extent, the "descent" of the testis. The testis usually enters the internal ring toward the end of the fifth month and appears at the external ring shortly thereafter. By the end of the sixth month it has reached the scrotum, but it is still embryonic in form, being cylindrically elongated, with the epididymis somewhat detached. The fetus is so small at this time that the length of the inguinal canal and the distance from the external ring to the scrotum are small. It is hard to tell how much the changes in position are due to actual migration of the testis and how much they are the result of differential growth of adjoining structures. In this descent, the left testis is usually a little in advance of the right.

The pathway which the processus vaginalis and testis will take is laid down very early in the embryo by the gubernaculum. Grossly, in unfixed material, the gubernaculum is a cord of edematous or fluid-filled tissue, which would seem to offer a pathway of little or no resistance. Outside the external inguinal ring this frays out into several strands (as has been often described), the largest going to the scrotum. When a testis occupies an unusual position, it is believed to have followed one of the minor attachments of the gubernaculum rather than the major one to the scrotum.

The most important observation in this part of our study was that the processus vaginalis and the testis always move together. In migrating from abdomen to scrotum, the testis was always within a few millimeters of the sac-like processus, and usually the structures were in close juxtaposition. Hence, anything that would block the evagination of the processus vaginalis would also block the descent of the testis and cause its arrest at that point.

Undescended Testis Is an Embryologic Failure

It is worth reporting that when a testis is seen at the operating table, it is almost always possible to state, entirely apart from its position, whether it has descended normally or is an undescended testis.

The configuration of the hernial sac and its attachments to the spermatic cord are quite unique in the undescended testis and do not resemble the normal testes seen in the corresponding age groups in the course of performing indirect inguinal herniorrhaphies. When found with an undescended testis, the hernial sac is extremely thin, is intimately attached to the cord structures, from which it can be separated only with difficulty, and is in such relation to the testis that it appears as though the gonad is within the hernia sac. Actually, the gonad is retroperitoneal, but is separated from the cavity of the sac by a membrane a few cells and about 400μ thick. Furthermore, the opening into the sac at the internal ring is usually slit-like, and the remainder of the sac widens rapidly, much as the mouth of a funnel (Fig. 5).

The testis itself, when undescended, is not like the normally descended testis. The shape is frequently globular, or sometimes very much elongated; the epididymis is widely separated instead of being attached to the long axis of the testis anteriorly. A Müllerian-duct remnant is almost always present on the undescended testis as an appendix testis, and the tunica albuginea of the undescended testis is not under the same tension as it is in the normal. It appears, then, that the clinical picture of an undescended testis found in the inguinal canal closely resembles the appearance of the descending testis of a fetus of 5-6 months' gestation, proving beyond doubt that the undescended testis is an embryonic arrest in development.

At the time of operation, it can be demonstrated clearly in most patients that there is a firm, fibrous barrier between inguinal canal and scrotum. Figure 6 illustrates the exploring finger at the lowest point of attachment of the processus vaginalis. Whether this barrier represents an embryonic struc-

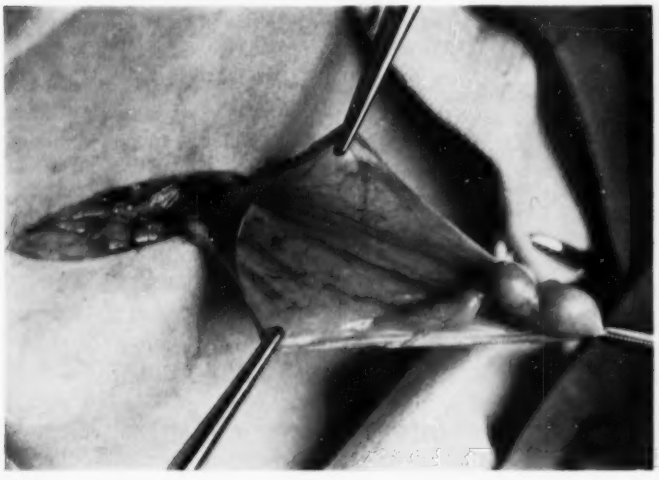

Fig. 5.—Funnel-like hernial sac stretched taut. The testis is seen to be elongated, the epididymis separate, and the appendix testis prominent.

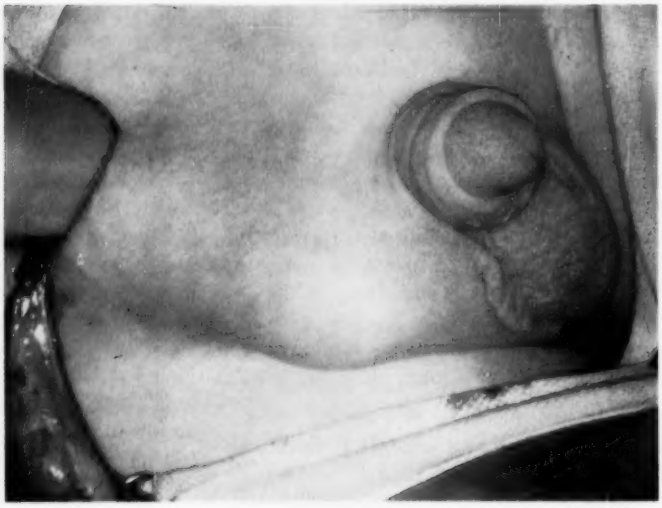

Fig. 6.—Finger exploring canal, seeking entry to scrotum. Obstruction to passage of finger represents lowest point of descent of embryonic processus vaginalis.

ture so firm that it could not be penetrated by the descending processus vaginalis, or whether the gubernaculum failed to guide the descending processus and testis through the normal penetration of this barrier is not known. The existence of this barrier is the best argument against the use of hormonal therapy to bring down an undescended testis.

Indications for Operation

The empty scrotum is also significant because it represents a series of indications for operation. Some of these indications are definite; others are open to question in the minds of some authors.

When a child has bilateral undescended testis, the primary indication is to provide for his fertility if at all possible. This can best be accomplished by bilateral orchiopexy, the two sides being done separately. The recent report of Drake [7] in which he reviews the reported surgical results of several surgeons is needlessly discouraging as to the results to be expected from surgery. Actually, Drake's review is a condemnation of the Torek operation and should not be leveled at orchiopexy in general. The good results of MacCullom [8] and of Gross and Jewett,[3] which Drake attributes to experience, we believe are being duplicated by other surgeons who are not depriving the testis of blood supply by the operative procedure itself. Drake's high percentage of testes which descend spontaneously would seem to indicate a series of predominantly retracted testes and not undescended testes.

In the unilateral undescended testis where fertility is not at stake, the most urgent indication for surgery is a psychological one, coupled, of course, with those given above. The boy with one testis is the butt of many jibes, and with the jokes collects a mass of misinformation about his virility, sexual development, and general future as a man which may lead to serious emotional problems.

The presence of a hernia would seem to be a definite indication for surgery, and at the same time orchiopexy could be carried out. That the inguinal testis is subject to injury is well known and also provides a reasonable indication for operation in which orchiopexy is carried out if possible. The management of the abdominal testis or the testis with too short a blood supply to bring it into the scrotum is the subject of another communication.[6]

No discussion of surgical indications is complete without reference to malignant change in the undescended gonad. Not know-

ing which statistics to believe, we take the point of view that until someone demonstrates without question that there is not an increased incidence of malignant change in the undescended testis, we shall continue to add it to the other indications for orchiopexy, but in a minor role.

Finally, if the operation for orchiopexy is one which preserves the function of the testis, a second gonad will be available should the other be injured.

Nothing has been included in this report concerning the administration of hormones to patients with undescended testis because we believe they have no place in the management of such patients. In another communication, the failure of hormonal therapy and its possible dangers will be discussed.[9]

Summary

There are several explanations for an empty scrotum. When a testis is palpable out of the scrotum, the differential diagnosis of retracted testis verus undescended testis must be considered. The patient with a retracted testis is not a candidate for either medical or surgical therapy. The patient with an undescended testis can be helped only surgically.

The descent of the testis is accomplished by a series of embryonic changes which are completed before birth. An undescended testis is a congenital defect representing an arrest in fetal development at about 20-24 weeks.

The prime indication for surgery in the patient with an empty scrotum bilaterally is to prevent certain sterility. Added to this are the indications for the correction of a unilateral defect which include psychological factors, prevention of injury to the testis, correction of a concomitant hernia, the possibility of eventual maligant change, and the possible functional usefulness of a second gonad.

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Observations on Undescended Testes

II. The Technique of Surgical Management

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The significance of the empty scrotum was the subject of a previous communication.[1] It was demonstrated that an undescending testis represents an embryologic arrest in development and that there is no more reason to expect spontaneous cure or correction by medical therapy than in any other congenital anatomical defect.

This report deals with our experience with an operative procedure, quite simple in technique, and productive of good cosmetic results. It has not been used long enough to evaluate from the standpoint of testicular function, but, piecing together bits of evidence from several sources, it seems reasonable to assume satisfactory functional results eventually in view of the size, shape, position, and consistency of the testis postoperatively.

Aims of Orchiopexy

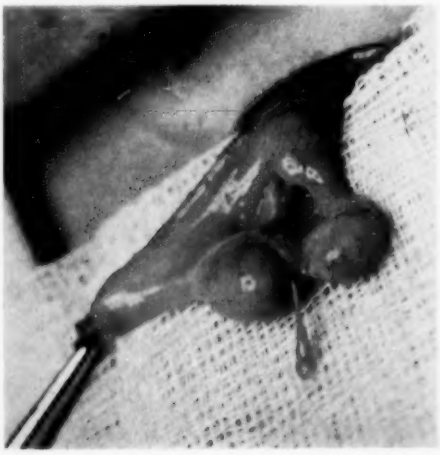
The goal of any operation for undescended testis should be a spermatogenic gonad in a cosmetically acceptable scrotal position. Such an operation should include a satisfactory method of dealing with a concomitant indirect inguinal hernia. Although any procedure which achieves these ends is justifiable no matter how long, complicated, or tedious, if the same ends can be attained by a simple, uncomplicated procedure, requiring little or no after-care, so much the better.

The final assessment of any operation for orchiopexy must be in the production of children by a patient who formerly had bilateral undescended testes. Testicular biopsies or sperm counts along the way may be indicative but not definitive. We acknowledge that our observations, chiefly in young patients, have not been of long enough duration to carry them into the reproductive age. However, being familiar with the operative technique reported by Gross and Jewett [2] to produce a fertility rate of 79%, we believe the operative procedure here described preserves the vital cord structures but uses a simpler technique, permitting of earlier ambulation and shorter hospitalization.

Anatomical Considerations

The undescended testis may be in the retroperitoneal space, just within the internal inguinal ring, in the inguinal canal, at the entrance to the scrotum, or in a position ectopic to the usual line of descent. The undescended testis is most frequently found

Fig. 1.—Undescended globular testis lying "in the sac" of an inguinal hernia. The epididymis is separate from the testis and unusually large. The appendix testis is prominent.



From the Surgical Clinic of the Children's Hospital of Philadelphia and the Harrison Department of Surgical Research, the University of Pennsylvania School of Medicine.

Fig. 2.—Dissection of a fetus of 24 weeks' gestation, illustrating limit of descent of processus vaginalis, with elongated testis lying "in the sac," a common finding in undescended testis lying in inguinal canal.

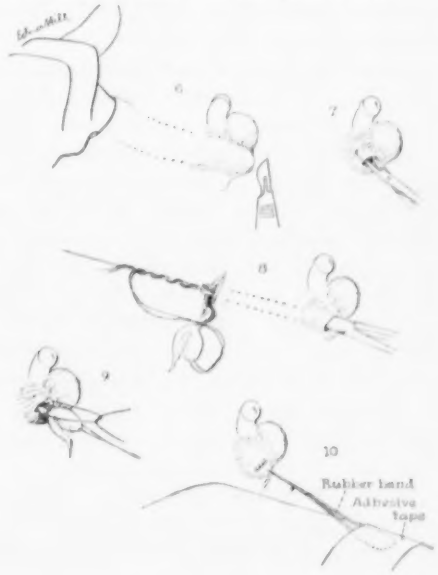
in the inguinal canal, and is usually attached securely to the medial side of the canal near the pubic tubercle.

There is usually no extension of the inguinal canal into the scrotum; rather, a fibrous barrier separates the two structures.

The undescended testis is frequently elongated or globular with a loose tunica albuginea rather than being ovoid and tightly encased by the tunica. The epididymis lies loosely attached to the testis and is usually longer than the gonad itself. The appendix testis, a Müllerian-duct remnant, is frequently prominent (Fig. 1).

Fig. 3.—Ligation of hernial sac and perforation of inguinal barrier to scrotum. *1, 2,* teasing hernial sac from cord; *3,* sac ligated, cremasteric-fascia dissection; *4, 5,* establishing inguinal tunnel to scrotum.

Fig. 4.—Preparation of subcutaneous pouch and fixation of testis. *6,* incising skin of scrotum over finger; *7,* separation of skin from subcutaneous tissue with small hemostat; *8,* silk suture through testis being passed through inguinal tunnel; *9,* suturing rent in subcutaneous tissue. *10,* suture passed through skin posterior to incision is fixed to thigh with rubber band and adhesive tape.



The position and appearance of the undescended testis resemble most closely the situation in fetuses of 20-24 weeks' gestation, as described by Hutchinson [3] (Fig. 2).

The blood supply is commonly shorter than will permit of the testis being placed in its normal position. The vas deferens rarely has been found to be too short. Retroperitoneally, the spermatic artery is usually in a straight line, while in the inguinal canal both vessels and vas deferens may be held in tight folds by the cremasteric fascia. These latter characteristics of the anomaly and the configuration of the hernia sac dictate the operative procedure we use (Figs. 3 and 4).

Operative Procedure

A transverse skin incision 1¼ to 1½ in. (32 to 38 mm.) long is made in a natural skin fold between the pubic tubercle and the anterior superior iliac spine, and is placed laterally over the inguinal canal. The full extent of the incision is used, cutting the subcutaneous fascia and adipose tissue as far as the ends of the wound.

The external oblique aponeurosis is split in line with its fibers, and the external ring is opened. The underside of the lateral flap of the external oblique is cleaned by blunt dissection almost to the shelving edge of Poupart's ligament. The cremasteric muscle fibers are identified, and a closed hemostat is inserted between the muscle bundles and then opened to expose the spermatic cord. The cord is lifted from the canal by passing a curved hemostat beneath it. Traction cephalad reveals that the cord is anchored to a point on the medial side of the canal in the region of the pubic tubercle. This attachment cannot be seen through the incision. It is not necessary to see it in order to divide it by sharp or blunt dissection.

The testis appears to be within the sac, which is then opened between two hemostats. The testis pops out of the apparent enclosure, and its tunica albuginea is grasped at the lower pole by a hemostat. The sac is slit cephalad to within 1 cm. of the internal ring. Hemostats then grasp opposite edges of the sac and stretch it taut, like a fireman's net. The operator holds the hemostat on his side in his left hand and, using curved, fine, blunt scissors, begins dissecting the cord structures away from the sac by looking through the transparent membrane. The vessels come loose more readily than the vas deferens, which is firmly attached. If it is possible to slide the blunt scissors all the way across the opened sac, this is done. If not, as is usually the case because of the comparatively voluminous sac, the dissection is carried as far as possible, and then the membrane is cut.

The posterior leaf of the sac just formed is then clamped to the anterior leaf previously made by opening the sac. This gives greater strength to the sac by doubling the thickness and permits one to continue the dissection without having to hold the lateral portion of the posterior sac to protect it from tearing.

Dissection is then continued in stages described above until the entire sac is free from the cord, and anterior and posterior leaves are clamped together. The cord is separated into the retroperitoneal space to a point slightly above the internal ring, where the vas can be seen to curve medially away from the blood supply. A simple, nontraumatic means whereby this may be accomplished is to place a closed thumb forceps between cord and sac and slowly let its natural spring open the forceps. In this way, nothing need be grasped, and injury to the cord is avoided. Great care must be taken to prevent tearing the sac because it is very fragile, and, once torn, is not easily picked up again.

With the sac freed from the surrounding structures, it is doubly ligated with sutures of cotton and the second stitch retained as a retractor and to be used later for fixing the sac beneath the internal oblique muscle.

At this point the cord is already found to be longer than when first encountered because of the transection of the hernial sac. The rest of the sac is slowly and carefully

peeled off distally, without difficulty and without bleeding from the cord. This maneuver gives still more length. It is now evident that the vas is long enough, but that the vessels, although elastic, usually will not permit the testis to be deposited in the low scrotum.

There now follows a painstaking dissection of the cremasteric fascia from the vascular bundles of the cord. This is begun by spreading the cord structures over the left index finger and slitting the cremasteric fascia longitudinally. As small bits of fascia are snipped away from vas and vessels, the cord becomes progressively longer. Neither the vessels nor the vas need be grasped with forceps. In practically every young patient, this dissection permits the placement of the testis in the low scrotum. In general, the older the patient the shorter the blood supply in comparison with the length needed, and the less rewarding is the dissection of the cremasteric fascia.

When the cord has been dissected, attention is turned to the inguinal canal. Exploration usually reveals a fibrous barrier between the canal and the scrotum, which sometimes can be pushed through by the finger and on other occasions must be pierced by an instrument to permit the probing finger to dilate the opening. The finger is pushed to the bottom of the scrotum and a small, 1 cm. transverse incision is made on the anterior scrotal skin over the contained finger. A pouch is developed just beneath the skin of the scrotum through this incision. A Kelly hemostat is then passed retrograde through the scrotal incision and guided into the inguinal canal by the exploring finger from above.

A single strand of 0 silk is then passed through the testis at the junction of the lower and middle thirds, care being taken to place it so that it will not tear the epididymis if it pulls through. The two ends of the suture are then placed in the same eye of the needle, which is then grasped by the Kelly hemostat and gently pulled so that the testis traverses the canal and scrotum and appears through the skin incision in the scrotum.

A single suture of fine cotton is taken in the subcutaneous layers of the scrotum at the point where the cord passes through them. The needle, with attached silk, is passed through the incision and out the dependent apex of the scrotum. The testis is thereby pulled back into a subcutaneous position in the lower scrotum. After this the scrotum is closed with four interrupted simple 000 plain absorbable surgical (gut) sutures on an Atraumatic needle.

Finally, the hernial sac is fixed to the underside of the internal oblique muscle and the wound closed in layers with nonabsorbable sutures, leaving the cord buried in the depths of the canal. The skin edges are approximated with four subcuticular sutures of 00000 white silk, and collodion is applied to the wound instead of a dressing.

Attention is then turned to one of the most important aspects of the procedure, the attachment of the suture in the testis to the thigh. The two silk threads leaving the scrotum are tied over a small roll of surgical gauze packing, which snugs the scrotal skin up to the testis. The silks are then tied to a rubber band, an old cuff of surgeon's glove being ideal for this purpose. This, in turn, is fixed to the thigh with adhesive tape so that there is no tension when the leg is straight.

Rationale of the Procedure

Any operation for correction of undescended testis which relies upon suture fixation to the thigh to give length to the cord, or even to maintain it, is doomed to failure because the inevitable tension cuts off arterial blood supply and results in atrophy and degenerative changes in the testis. The fascial fixation advocated by Torek [4] and by Wangensteen [5] is safe in regard to preservation of the testis only when the cord is excessively long, and when this is true, there is no need for such fixation.

The designation of a "traction suture" in orchiopexy is a misnomer. The elastic fixa-

tion described by Cabot and Nesbit [6] was a real contribution to the surgery for undescended testis. The situation is one of having to place a raw spermatic cord through a raw inguinal tunnel. The objective is to maintain the testis in optimum position while adhesions form. Many orchiopexies fail after good dissection and proper placement of the testis in the scrotum because adhesions form in the position to which the elastic blood supply retracts the testis. In the operation here described, great care is taken to apply the rubber band so that it is under no tension, but merely taut, when the legs are side by side and fully extended.

This operation relies primarily on dissection of the cremasteric fascia to give length to the cord. So much can be achieved by this dissection carefully done that high retroperitoneal dissection, such as that advocated by Gross and Jewett,[2] is unnecessary except in rare circumstances. It has been our experience that high retroperitoneal dissection gives little in length to the blood supply when compared with equal time spent on cremasteric-fascia dissection. Nor have we found useful, except on rare occasions, the modification of Lewis,[7] in which the deep epigastric vessels are cut to allow greater length to the cord.

Use of either high retroperitoneal dissection or division of the deep epigastric vessels not only eliminates unnecessary dissection but saves time and permits the use of a small incision.

The reason we open the scrotum and pass the testis through the opening before placing it in the subcutaneous pouch is that this is the only way in which we can be sure that a cavity has been made in the scrotum all the way to the bottom. We experienced many failures before using this technique, when we merely pushed the testis into the scrotum as far as we could and then passed the sutures blindly through the scrotal skin. The creation of the subcutaneous pouch provides an additional safety factor, it seems to us, in placing the testis as close to the bottom of the scrotum as is possible.

The Short Cord and the Abdominal Testis

When the spermatic cord is so short that any procedure designed to give length to the cord is insufficient to achieve the desired result, one is faced with the decision whether to leave the gonad as far down as it will come or to extirpate it. It has been our custom to extirpate if the undescended testis is unilateral. If the youngster has bilateral undescended testes, we then bring each testis as far down as it will come and leave it there. We inform the parents of the possibility of malignant change but minimize its dangers. If one of bilateral undescended testes can be brought down, we extirpate the other.

When a testis is truly abdominal, it usually has so short a cord that a scrotal placement is an impossibility. If such a gonad can be brought into a palpable position in the canal, this is done on one or both sides. If the gonad cannot be brought into the canal, we extirpate both testes after thorough discussion with the parents and after having obtained written permission. We believe that until it is proved that there is no increased incidence of malignant degeneration in testes which remain undescended, we would be wrong to leave a potentially dangerous gonad in a position where changes in size could not be detected. Under these circumstances, puberty would have to be controlled artificially.

Rarely one finds no cord in the canal and no abdominal testis. It is our custom then to slide the skin incision cephalad, make a low muscle-splitting incision into the lower abdominal wall without entering the peritoneal cavity, and explore the retroperitoneal space down to the seminal vesicles. If no vas deferens is found, we cease our search. If a vas deferens is found that leads to no testis, we look no further. If a testis is found, it is dealt with according to principles laid down previously.

Unilateral Versus Bilateral Undescended Testis

Believing that bilateral undescended testes in practically every instance are the chance occurrence of two unilateral lesions in the same patient, we consider bilateral defects to be amenable only to surgical correction. In bilateral lesions, it is our custom to separate the procedures by at least one month. The conscious and unconscious splinting the patient maintains following a unilateral operation is impossible when the two sides are done simultaneously, and one side suffers as to end-results. We do the left first because the left cord is usually longer.

If the scrotum is empty bilaterally and a gonad is felt only on one side, we do the contralateral side first. In this way, if no gonad is found or if orchiopexy is not possible, we can do the other side at the same time without jeopardizing the result of either side.

Effect of Age on Ease of Operation and on Result

There is no doubt that the spermatic cord is proportionately longer in the younger patient; therefore, the likelihood of getting a testis low in the scrotum is better in young children. However, to offset this advantage, there is the fact that in no surgical procedure is there a more flimsy membrane than the hernia sac associated with an infant's undescended testis. Although we acknowledge the difficulties of operating for undescended testis in the very young, we believe that in experienced hands the good results make the necessary extra care seem worth while.

The age of election should be based on at least two factors: (*a*) the cosmetic result obtained at various ages, and (*b*) the age under which a functioning testis may be expected after placing it in the scrotum.

We have already indicated that the technique presented here is the best in our hands of any we have tried for any age and that the best cosmetic results have been in the young. It is our own custom to do an orchiopexy anytime we encounter an undescended testis after the age of 6 months. We do not advocate this for the surgeon who has no continuing experience with the procedure.

The age of election in reference to obtaining a functioning testis is the object of a study in this clinic based upon biopsies before and after orchiopexy at various ages. On the basis of an incomplete study, we believe that orchiopexy should be done before the age of 6 years.[8] It is quite possible that further study will indicate that this age should be closer to infancy.

The Second Attempt at Orchiopexy

One of the reasons we are pleased with the technique described here is that we have used it as a second procedure on patients operated upon previously by others, and previously by ourselves using other techniques. In many patients we have been able to reclaim a testis from the canal or bring one of high scrotal position into the low scrotum. We do not believe that a second operation for orchiopexy can be expected to improve the position of the testis if the first operation is done by the technique described here because it would not yield enough additional length to the cord to be worth while.

Postoperative Care

Postoperatively, the child may be as active as he wishes. Infants may be held in the mother's arms as soon as they react from anesthesia. Children old enough to walk are made to do so no later than 24 hours postoperatively. Discharge from the hospital takes place on the day following surgery, and most hospitalizations are of 44 hours' duration. The only restriction on activity is bathing, to protect the adhesive holding the elastic band. The silk suture through the scrotum is removed one week postoperatively by cutting one strand and pulling

on the other, unless it comes out spontaneously before that time. The sutures closing the inguinal incision are subcuticular and are not removed.

Results

Our experience covers more than 750 orchiopexies done by several procedures, including that of Gross and Jewett and the one reported here. No Torek procedure has been done in this series.

As has been stated, our results at the present time can be judged only by cosmetic appearance and palpation of the testis. Our patients are not old enough to evaluate spermatogenically, and because so many of them are young children, they will not be ready for this type of study for years to come.

We have been pleased to note how many abnormal-appearing testes placed in the scrotum could not be differentiated from the contralateral mate four months postoperatively. The normal rubbery consistency which many of these testes exhibit after the scrotal induration has subsided and the fact that they feel just like their mates lead us to believe that they have not been damaged in the procedure.

Some testes which are smaller than their mates grow in size but never quite catch up. Others seem to have acceleration in growth, especially those placed in the scrotum in early life.

In a recent representative series of patients being studied on the basis of testicular biopsy, the clinical results were also tabulated. Of those patients in whom any attempt at orchiopexy by this procedure was possible, 85% had a good result. Of the remaining 15%, half had fair and half poor results.

Comment

It is discouraging to read reports, such as that of Charny,[9] concluding that there are no satisfactory techniques for orchiopexy, or to read the conclusions of Kimbrough and Reed,[10] who state, after describing a method of orchiopexy, that fertility will rarely be achieved in bilateral undescended testes. These authors and others, such as Drake,[11] who reviewed the discouraging results of a number of surgeons, are directing criticism at techniques of orchiopexy, many of which use fixation of the testis to the fascia of the thigh and thereby compromise blood supply by tension.

Charny's [12] study of 145 biopsies of adult testes brought into the scrotum before the age of 16 is a valuable one. Most of the patients were not his own, and the operations had been done by a variety of techniques. None showed spermatogenesis which could be considered adequate for fertility. At first appraisal, this may seem to be most discouraging. However, many of the operations were of the Torek variety, and spermatogenesis is not to be expected. Others may have been done too late in life to expect spermatogenesis. Finally, some men seeking help for sterility may have had a defect in spermatogenesis that precluded a successful outcome from orchiopexy by any technique.

It may well be that histologic study of young testes is an inadequate means of determining the age for orchiopexy. It has been shown that orchiopexy for bilateral undescended testes can produce fertility. Our experience clearly shows that a low scrotal position for the testis is more easily achieved in the very young. In addition to this indication for early operation, we cannot help believing that, inasmuch as undescended testis is an embryologic failure, the best functional result might be obtained when the testis is put where it belongs as close to the embryologic age as feasible.

Summary

True undescended testes are embryologic failures and therefore are congenital defects amenable only to surgical correction. A technique of orchiopexy is described the essential features of which include dissection of the cremasteric fascia to achieve cord length, the creation of a subcutaneous pouch in the scrotum for the testis, and the maintenance of the testis in the lowest scrotal

position until adhesions form. The operation is possible through a small incision, requires less than two days' hospitalization, and achieves satisfactory results based upon cosmetic appearance and testicular palpation.

Several reasons for performing orchiopexy in infants and young children are discussed.

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Hirschsprung's Disease

A Lethal Problem in Early Infancy

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Hirschsprung's disease is particularly devastating to children under 6 months of age, and, contrary to popular opinion, relatively few survive early infancy to develop eventually the classical and better-known clinical picture originally described by Hirschsprung.[1] Even since 1948, when Swenson and Bill published their original work on the development of a curative surgical operation to correct this anomaly,[2] the high mortality among young infants with this disease has continued as a shocking paradox to the many subsequent glowing reports on the successful management of this problem in slightly older children.

The term Hirschsprung's disease implies herein the specific functional disorder of bowel caused by congenital dysplasia of the myenteric nervous plexus. The indefinite term megacolon is used only in its literal sense, meaning an enlarged and hypertrophied colon. Idiopathic megacolon, as defined by Bodian, Stephens, and Ward,[3] and megacolon due to a distal organic obstructive lesion will not be discussed in this paper.

Prior to 1954, Klein and Scarborough [4] collected 50 cases of Hirschsprung's disease in newborn infants and reported 70% fatalities. Since 1947 I have found reports in the English-recorded literature totaling 156 proved cases in infants under 6 months of age, of whom 76 (48.7%) succumbed. In this latter group the mortality might have been closer to 64% had not 24 of the 80 survivors been successfully relieved of their morbid symptoms by surgical creation of a proximal enterostomy. These startling losses reflect a common failure of recognition of this serious problem during neonatal life, and, thereby lacking proper therapy, many infants have been needlessly lost to lethal complications which frequently compound this disease.

TABLE 1.—*Hirschsprung's Disease in Infants Under Six Months of Age Reported Since 1947*

Author	Date	No. of Cases	Method of Treatment	Alive	Dead
Santulli	1956	14	Conservative	6	8
		15	Colostomy	15	
Keefer & Mokrohisky	1954	9	Conservative enterostomy or resection	6	3
Bodian, Stephens, & Ward	1949	28	Conservative	23	5
Klein & Scarborough	1954	2	Explored only		2
		3	Enterostomy, in 1 too far distal	2	1
Burnard	1950	7	Conservative	5	2
Burnard (collected)		23	Conservative	5	18
Bodian, Carter, & Ward	1951	1	Conservative (whole intestine)		1
Zuelzer & Wilson	1948	2	Colostomy	2	
		9	Conservative		9
Bowden, Goodfellow & Munn	1957	2	Explored only		
		1	Ileoascending colostomy		5
		2	Conservative		
Stockdale & Miller	1957	2	Colostomy	2	
Goldbloom & Wiglesworth	1954	3	Conservative		3
Potts, Boggs & White	1952	2	Explored only		2
		5	Colostomy	3	2
		2	Colostomy too distal		2
		1	Resection, pull-through	1	
McDonald & Evans	1954	5	Colostomy	1	4
		1	Ileostomy		1
		5	Conservative	3	2
Ehrenpreis	1955	10	Conservative	5	5
Hallenbeck & Waugh	1952	1	Colostomy	1	
Total		156		80 (51.3%)	76 (48.7%)

TABLE 2.—*Experience with Hirschsprung's Disease in Infants Under Six Months of Age, 1954-1957*

Case	Age	Race	Sex	Symptoms	Method of Diagnosis	Operation	Age of Each	Result	Segment Aganglionic	Radiological Findings
1	3 wk.	W	F	Persistent low bowel obstruction	Exploratory lap.; frozen section	Transverse colost. Term. ileostomy Ileoproctostomy	3 wk. 4 wk. 18 mo.	Unimproved Improved Cured	Entire colon & 8 in. ileum	B. E.* called normal; plain films showed small-bowel distention
2	48 hr.	W	F	Acute lower bowel obstruction	Clinical diag.; explor. lap.	Transverse colost.	48 hr.	Improved	Sigmoid & rectum	B. E. called normal; large & small bowel distended
3	6 wk.	C	M	Recurrent low bowel obstruction	Clinical diag.; repeated biopsies	Transverse colost. Coloprotostomy	6 wk. 1 yr.	Improved Cured	Rectosigmoid	B. E. normal 3 times before 6 wk. of age
4	6 wk.	W	M	Peritonitis	Autopsy	None	6 wk.	Died	Rectosigmoid	No radiographs
5	6 wk.	C	M	Recurrent low bowel obstruction	Clinical diag.; suggestive Biop.	Transverse colost. Coloproctostomy	6 wk. 1 yr.	Improved Cured	Rectosigmoid	B. E. normal 3 times before 6 wk. of age
Sibs 6	15 wk.	C	M	Constipation & diarrhea; wt. loss	Barium enema	Sigmoid colostomy	15 wk.	Improved	Rectosigmoid	B. E. normal @ 5 days B. E. diagnostic @ 13 wk.
7	3 mo.	W	M	Diarrhea, fever, obstipation	Autopsy	Rt. hemicolectomy & ileocolostomy	3 mo.	Died	Rectum & sigmoid	B. E. & G. I. series called normal @ 6 wk
8	2 mo.	W	M	Constipation & diarrhea; malnutrition	Barium enema	Sigmoid colostomy Coloproctostomy	2 mo. 1 yr.	Improved Cured	Rectosigmoid	B. E. normal @ 1 mo. of age B. E. diagnostic @ 2 mo.
9	24 hr.	W	M	Acute small bowel obstruction	Laparotomy	Ileostomy	24 hr.	Improved	Entire colon & 18 in. ileum	No biopsy done; small bowel obstruction
10	4 days	W	M	Acute low bowel obstruction	Barium enema	Sigmoid colostomy	5 days	Improved	Rectosigmoid	B. E. diagnostic, 4th day of live
11	9 days	W	M	Distention, emeses, bloody diarrhea	Clinical diag.	None to date		Living; unhealthy	Rectosigmoid, ? more (?)	B. E. @ 2 wk. very suggestive; not typical
12	36 hr.	C	F	Acute lower ileal obstruction	Barium enema & laparotomy	Ileostomy & right hemicolectomy	36 hr.	Improved	Entire colon & 18 in. ileum	B. E. shows microcolon & terminal ileum
13	3 days	C	F	Acute lower ileal obstruction	Barium enema & laparotomy	Ileostomy & right hemicolectomy	4 days	Died 24 hr. P. O.	Entire colon & 18 in. ileum	B. E. shows microcolon & terminal ileum

* B. E. = barium enema.

It is the purpose of this presentation to highlight the earliest manifestations of Hirschsprung's disease in very young infants, in contrast to the classical syndrome better recognized in somewhat older children, and to recommend urgently an aggressive program of surgical management which should effect a considerable decrease in mortality among infants suffering the effects of this anomaly.

Historical Review

Hirschsprung's original observations, reported in 1886, were made on two baby boys, aged 7 and 11 months, who had suffered unrelenting symptoms of chronic constipation, malnutrition, and intermittent episodes of intestinal obstruction since birth. Progressive enlargement of the abdomen continued until their deaths, when a tremendous, hypertrophied, feces-packed colon was noted at autopsy in each child.

Hirschsprung's descriptive term, "congenital hypertrophic dilatation of the colon," erroneously fixed attention upon the megacolon as the primary site and cause of the disease, and during the ensuing 60 years little progress in true understanding of the problem was made, until investigators directed their studies to the relatively normal-appearing bowel distal to the megacolon.

In 1946, Swenson, Rheinlander, and Diamond ingeniously demonstrated the consistent failure of coordinated propulsive peristaltic activity throughout the segment of bowel distal to the megacolon,[5] an effect which imposes a variable degree of obstruction to fecal flow. The primary pathological lesion responsible for this unphysiologic phenomenon has been confirmed by several noted investigators as a congenital developmental anomaly characterized by the absence of parasympathetic ganglion cells and an increased incidence of nonmyelinated nerve fibers within the myenteric nervous plexus of the aperistaltic distal segment of bowel.[6-9]

Ehrenpreis, in 1946, was first to note the consistent absence of a megacolon in 10 symptomatic newborn infants who within a few months did develop this characteristic feature, as well as other classical manifestations previously described by Hirschsprung. While establishing the fact that the megacolon per se is not present at birth, he observed several other important features in the disease pattern at this age which differ essentially from the classical picture in somewhat older children.[10]

Pathological Physiology

Though the primary pathological lesion in all victims of Hirschsprung's disease is fundamentally the same regardless of age, the severity of the disease may vary extremely from patient to patient, and even within the same patient from time to time. The type and severity of symptoms manifest are governed by a strategic combination of circumstances affected by the degree of compensatory hypertrophy and dilatation of the proximal bowel, which is capable of normal peristalsis, the degree of inspissation and impaction of feces within the proximal and distal segments, and the degree of obstruction imposed by the aperistaltic distal intestine.

The most frequent manifestation of Hirschsprung's disease in a newborn child is acute intestinal obstruction, with gaseous abdominal distention, hyperactive peristalsis, meconium or fecal vomiting, and scanty-to-absent passage of meconium stools during the first few days of life.[4,8,10] Less commonly, a profuse and malodorous watery diarrhea may dominate the picture.[20] Digital rectal examination usually reveals an abnormally tight and empty rectum, often with an easily dilatable stenosis at the junction of rectum with sigmoid colon. Following such dilatation, or upon evacuation of an enema, the apparent symptoms of obstruction may be completely relieved, only to recur in time after a variable and unpredictable period of seeming good health.

In the average infant afflicted with this anomaly the entire bowel is essentially normal in size and muscular development at the

time of birth; yet its propulsive motor power may be insufficient to overcome the obstructing resistance imposed by the distal aperistaltic segment. When swallowed air and fluids fill the entire proximal intestine, vomiting of bowel contents results unless the distal obstruction is relieved. With some passage of time the proximal bowel hypertrophies to compensate for its added work load, while gradual dilatation of the same segment occurs simultaneously to accommodate the increasing accumulation of inspissated stool. Development of a megacolon creates an elastic reservoir which can accommodate tremendous quantities of fluid, gas, and feces without causing retrogressive accumulation in the more proximal intestine. Hence, in children past the neonatal age the symptoms of chronic constipation, abdominal enlargement, and diminishing incidence of vomiting gradually replace the more dramatic symptoms of acute intestinal obstruction observed in younger infants.

With occurrence of chronic fecal stasis in the newborn child there is a rapid increase of bacterial mass and putrefaction of intestinal contents in a viscus not yet well accustomed to an unsterile medium. Accumulated inspissated feces may cause mechanical irritation and ulcerations in a delicate mucosa, creating penetrations which bacteria may readily invade during a period of low local resistance, even to organisms commonly regarded as nonpathogenic. Severe, and oftentimes lethal, complications in the form of nonspecific diarrhea, phlegmonous or ulcerative enterocolitis, septicemia, pylephlebitis, and peritonitis with or without gross perforation of the bowel have accounted for a majority of the deaths among these young infants who survived milder episodes of obstruction.[8,10,12,20,22]

Many of the milder cases of Hirschsprung's disease have responded rather well to conservative treatment with laxatives, stool softeners, enemas, and parasympathomimetic drugs, and many physicians advocate this means of therapy in every possible instance to avoid early surgical intervention. However, in a large proportion of the reported cases reviewed (48.7%) the infants succumbed to the lethal complications mentioned while receiving accepted medical therapy. One might assume that prevention of dramatic attacks of intestinal obstruction by conservative means during early infancy allows a false sense of security to prevail while subtler, but nevertheless potentially lethal, complications become established.

It would seem those children with the longest segments of aganglionic bowel should present the earliest and severest symptoms, but there has been no such absolute correlation among the collected cases, nor among those I have observed. Though most infants reported with a long segment involved presented severe, and often fatal, problems, there are many more with the commoner, short segment whose disease was equally severe or fatal. However, there are extremely few reported cases with the long-segment variety in which the children survived early infancy without surgical relief of their functional obstruction.

Radiological Features

When acute intestinal obstruction occurs with the commoner, short-segment variety, ordinary radiographs reveal diffuse fluid and gaseous distention of the entire large and small intestine, which should indicate an obstructing lesion in the lower bowel. Barium-enema examination reveals no evidence of an organic obstruction, and demonstration of an essentially normal colon is usually reported at this very early age. If the examination is done very carefully with a small, noninflatable catheter tip strapped just inside the anus, one might observe a relatively contracted segment of rectum or sigmoid colon as it is slowly filled under fluoroscopic observation, even though there is no megacolon present at this time. This feature may be even more clearly demonstrated with double-contrast technique, as suggested by Keefer and Mokrohisky,[11] or follow-up films made several hours after evacuation may show a more characteristic picture, with abnormal

retention of opaque medium in the bowel just proximal to the aganglionic segment.[12]

In an instance in which most of the colon, or an even longer segment of bowel, is aganglionic, ordinary radiographs may show a pattern of complete small-bowel obstruction; yet a completely patent, but abnormally small-caliber, colon may be outlined by barium enema.[8,13] Reflux of opaque medium into the dilated proximal intestine rules out an atresia and suggests either a mechanical or a functional failure of motility. If coordinated peristalsis is lacking, then the opaque medium will not be evacuated, and one may reasonably assume that the obstructive phenomenon is the manifestation of a neuromyenteric anomaly.

On rare occasions a newborn child may exhibit all mentioned evidences of lower-bowel obstruction with the actual cause a simple mucus or meconium cast obturating the distal colon. Simple enemas relieve this problem by assisting in passage of the characteristic mucosal-patterned cast which can be visualized as a filling defect in the colon upon careful barium-enema examination. In several such cases which I have observed further intestinal dysfunction has not occurred.

Once the typical megacolon has developed, barium-enema examination should demonstrate a distinct caliber change in the region of the junction of normal and aganglionic bowel. However, this characteristic may be easily overlooked or masked by improper technique of examination. The technical method described by Swenson and associates [14] in 1949 is applicable to patients of all ages, but the generally high percentage of diagnostic failures among young infants would indicate the need for further refinements in technique and more painstaking effort on the part of examiners. Reliable radiological diagnosis can be made with a rather high degree of regularity even in newborn infants,[11,12,15] but a failure to demonstrate the characteristic caliber change should by no means rule out the probability of Hirschsprung's disease in an infant whose clinical findings suggest this diagnosis. If there remains any doubt or indecision, the riddle may be solved by microscopic examination of a satisfactory rectal muscle biopsy specimen, as has been recommended by Swenson, Fisher, and MacMahon.[16]

Summary of Cases

My own experiences and observations during the past three years concern 13 infants, 4 girls and 9 boys less than 6 months of age, in 12 of whom histological proof of Hirschsprung's disease was obtained. The 11th case was diagnosed from clinical manifestations at 9 days of age, and by the age of 5 weeks the infant had developed a visible and palpable megacolon. At 4 months he continues to suffer intermittent episodes of abdominal distention, fever, and passage of dark, bloody stools, but to date his parents and pediatrician have refused permission for an enterostomy.

One infant, first seen at 6 weeks of age, promptly died with peritonitis resulting from a perforation in the sigmoid colon, found at autopsy, just proximal to the beginning of aganglionic bowel. One of three deaths occurring in this series was possibly preventable, that of a 3-month-old boy who developed phlegmonous ulcerative colitis with multiple colonic and appendiceal perforations while receiving conservative treatment. Though he was ultimately operated upon, he succumbed to peritonitis after a long sequence of further complications.

Among 12 infants examined by barium enema before 6 weeks of age, 8 were reported to have a normal colon; 3 showed a very small lumen throughout the entire colon, and in 1 a small, contracted rectosigmoid colon was carefully demonstrated on the fourth postnatal day.

The aganglionic segment included only the rectum or sigmoid colon in nine of this series, and in four the entire colon and terminal ileum was involved. In 6 of the living 10 infants a proximal enterostomy was established as a life-saving procedure for relief of acute or recurrent intestinal ob-

struction soon after or within six weeks of birth. In an infant 2 months of age, severe and progressive malnutrition associated with alternating constipation and watery diarrhea was relieved by a sigmoid colostomy. A prophylactic elective colostomy was made in a 5-day-old child following an episode of acute colonic obstruction and, again, in a 15-week-old sibling of another child in this series who had begun to lose weight and manifest mild fever with intermittent constipation and diarrhea. When a colostomy was made in two patients with diarrhea, the proximal colon appeared inflamed and moderately edematous, showing some degree of correlated enteritis in progress at the time of operation.

Of the 11 infants treated early by enterostomy, 10 improved dramatically and have done well to date. Six have later undergone definitive resection of the aganglionic bowel, with restoration of continuity by the Swenson procedure, including one girl in whom the entire colon and terminal 8 in. (20 cm.) of ileum was involved. Following ileoproctostomy at 18 months of age and 23 lb. (10,433 gm.) body weight, this child now appears in excellent health, at 3 years of age, and has had normal continence of feces since 2 years of age. Absence or reduced numbers of ganglion cells were found in the terminal 18 in. (45.5 cm.) of ileum and the entire colon of two girls and one boy born with atypical meconium ileus. An ileostomy was established in each case soon after birth. The two girls were born prematurely; one died 24 hours after ileostomy, and the other is making satisfactory progress, at 10 weeks of age. The boy is now 10 months of age and is scheduled soon to undergo a definitive ileoproctostomy.

In this series of 13 cases, there are two sole Negro male siblings with no previous known familial incidence, and there is one white boy in a family of three children whose sister and paternal male cousin are afflicted with Hirschsprung's disease. Bodian, Carter, and Ward summarized the literature prior to 1951 to point out the familial incidence of this disease; they estimated a 20% probability, with variation according to sex and previous incidence within the same generation.[17]

Surgical Management

Whenever abdominal exploration is undertaken during early infancy for relief of intestinal obstruction of unknown cause, the possibility of Hirschsprung's disease should be seriously considered until an unequivocal organic obstruction is found. Finding no such lesion, a disturbance of motility is a likely explanation, and the abdomen should not be closed until positive identification of normal ganglion cells has been made by competent frozen-section examination of a biopsy specimen of bowel wall taken from a point distal to the obviously distended intestine. If ganglion cells are lacking, creation of an enterostomy should be mandatory at a more proximal point, where normal myenteric histology can be demonstrated by the same microscopic technique. Definitive resection of the aganglionic bowel and restoration of continuity by the Swenson operation should not be attempted in small infants. Later closure of the enterostomy and definitive resection are best done when the child has reached some 20 lb. (9072 gm.) body weight and is in good general health.

Bodian, Carter, and Ward have estimated that some 90% of such patients have only their rectum or rectosigmoid colon affected, while the remaining 10% will be found with a longer segment, or possibly the entire intestine, involved.[8,17,22] A few rare instances of alternating normal and aganglionic segments have been reported,[11,18] but in the overwhelming majority no normal bowel exists between the most proximal beginning of the aganglionic lesion and the anus.

When the myenteric anomaly extends proximally into the ileum, one may find a picture quite similar to that of meconium ileus complicating cystic fibrosis of the pancreas. Differentiation of these etiologically separate entities may usually be made by gross characteristics of the meconium, but

a frozen-section examination for ganglion cells may again make the diagnosis certain when doubt exists. Proper surgical management of these more difficult cases leaves much to be desired in making the best of a bad situation. Unlike meconium ileus, the distal aganglionic bowel is incapable of normal peristaltic function, and restoration of continuity of this bowel would result in an anastomotic breakdown or further recurrence of obstruction. Though it is obviously undesirable, the immediate solution is to create an ileostomy at the most distal point in the small bowel where normal myenteric histology may be demonstrated. An infant can survive with an ileostomy, though the nursing care and nutritional problems are quite difficult until the child adapts to his situation. When the child attains sufficient size to permit a satisfactory technical procedure to be done, a pull-through type of ileoproctostomy may afford a gratifying result.

Summary and Conclusion

Analysis of all cases of Hirschsprung's disease reported reveals an appalling mortality in infants prior to 6 months of age, particularly among those receiving medical treatment alone. In 24 of 80 survivors of a group of 156 cases a proximal enterostomy was established prior to 3 months of age, with gratifying relief of intestinal obstruction or intractable diarrhea. This experience is similar to my own, more limited observations and has fostered the reasonable conclusion that many more afflicted babies would survive early infancy if all those recognized prior to 6 months of age were afforded similar surgical treatment as a routine policy of management. This should include all infants for whom the diagnosis has been established, even though their current status with conservative management seems to be quite satisfactory.

Early diagnosis is the greatest problem today precluding proper management and relief from Hirschsprung's disease during early infancy, when the clinical manifestations of this anomaly present a picture strikingly different from that originally described by Hirschsprung in somewhat older children. Radiological characteristics are likewise peculiar to this age and contrast sharply with the established criteria well recognized in older children.

A review of the literature since 1947 reveals a mortality in excess of 48% among infants under 6 months of age afflicted with this malady. Severe and potentially lethal complications frequently compound this problem in young infants, even while under treatment by accepted nonsurgical means.

Surgical creation of a proximal enterostomy is recommended for all afflicted infants under 6 months of age in order to avoid these serious complications and to improve upon this appalling mortality incidence.

Parents of a child with Hirschsprung's disease should be advised of the probability that future progeny of their union may also suffer this anomaly.

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Meckel's Diverticulum in Children

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The diverticular outpouching of the small intestine which bears the name of Johann Meckel was described in medical literature as early as the 17th century, but it remained for this German anatomist to give the first adequate description of the pathologic entity and to associate it with the faulty obliteration of the omphalomesenteric duct.

This diverticulum occurs relatively frequently and has even been claimed by some to be the commonest anomaly of the gastrointestinal tract.[1] The generally accepted incidence of 1% to 2% in the over-all population is comparable to the occurrence of inguinal hernias in childhood, which is, likewise, about 2%. Kittle, Jenkins, and Dragstedt,[2] found this lesion in 0.5% of a large autopsy series, while in the Children's Hospital of Pittsburgh the incidence has been 1.3% in an autopsy series of 1809 patients. This would seem to point up a relative frequency of occurrence of this lesion without clinical significance, but it is because of the frequency of serious pathologic changes that a Meckel's diverticulum becomes important from a clinical point of view.

Embryology and Pathologic Anatomy

Between the second and the fourth week of fetal life, the yolk sac is joined to the apex of a loop of primitive midgut and is referred to as either the omphalomesenteric duct or the vitellointestinal duct. Through this duct the embryo receives nourishment from the yolk sac until such time as the placenta takes over the nutritional needs of the fetus and the omphalomesenteric duct begins to be obliterated (Fig. 1). This obliteration is generally thought to be complete by the seventh week. Failure to become obliterated at some point along its course leads to a variety of anomalies, one of which is termed Meckel's diverticulum. The first abnormality of the omphalomesenteric obliteration may be a complete patency from the intestine to the umbilicus (Fig. 2*A*); this is rarely seen. A second abnormality may be a partial obliteration of the duct at the intestinal end, leaving some of the duct patent beneath the umbilicus (Fig. 2*B*); this occurs occasionally. Complete obliteration of the duct with maintenance of its attachment to the underside of the umbilicus is still another variety of abnormal obliteration (Fig. 2*C*); one author[3] places the incidence of this type at 10%. A fourth variety of anatomical aberration may be the obliteration of the duct at the umbilical and intestinal ends with persistence of its patency in the midportion (Fig. 2*D*); this is sometimes referred to as an omphalomesenteric cyst and occurs very infrequently. Finally, obliteration and elimination of the duct with the exception of the few centimeters at the intestinal end leaves one with the classic diverticulum of Meckel, which is present in over 80% of cases having omphalomesenteric duct abnormalities (Fig. 2*E*). The diverticulum in this case is antimesenteric and has its own blood supply, derived from the embryonic vitelline arteries, which become part of the superior mesenteric system. Meckel's diverticulum occurs most frequently in the 100 cm. proximal to the ileocecal valve, and this location accounts for over 90% of all diverticula.

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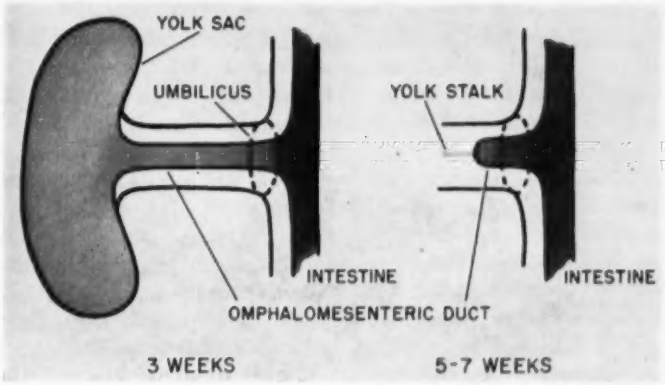

Fig. 1.—Embryology of omphalomesenteric duct.

Histology

At least one of the major clinical pictures caused by a Meckel's diverticulum is due to the fact that there is ectopic mucosa present in the outpouching. The presence of such aberrant tissue as gastric glands, pancreatic cells, etc., can be most easily explained by the fact that the omphalomesenteric duct consists of tissue which has totipotential cells within it. Although these disappear with the obliteration of the duct in a normal child, they may persist in a child who ultimately has a Meckel's diverticulum. It has been of interest to us to note that 29 of 50 resected specimens from symptomatic cases at our hospital have contained either gastric tissue or pancreatic tissue or a combination of the two.

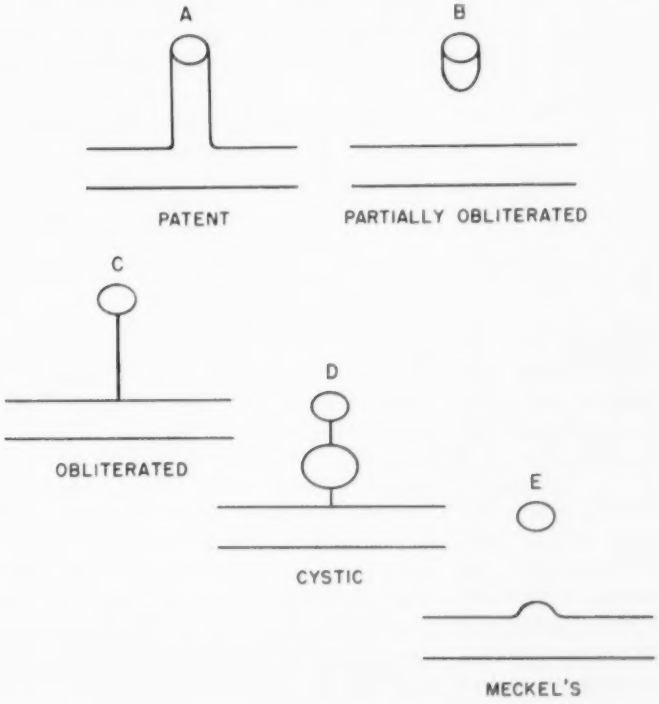

Fig. 2.—Abnormalities of the omphalomesenteric duct.

Clinical Types of Meckel's Diverticulum and Presenting Complaints

	Cases	Melena	Pain	Vomiting
I. With symptoms				
A. Ulceration	23			
Nonperforated		17	2	1
Perforated		1	1	1
B. Inflammation	14			
Acute		6	3	1
Chronic			3	
Perforated			1	
C. Obstruction	11			
Intussusception		1	1	4
Volvulus				3
Bands			1	1
D. Umbilical sinus	2			
	50			
II. Incidental				
A. At surgery	13			
B. At autopsy	21			
	34			
Total	84			

Clinical Pictures

The fact that a Meckel's diverticulum occurs in 1% to 2% of the pediatric population does not accurately portray the clinical significance of this lesion, because a certain percentage of such children will possess the lesion without ever having any symptoms from it. Over a 15-year period (1940-1955) at the Children's Hospital of Pittsburgh a total of 84 cases having such a diverticulum have been encountered at surgery or autopsy. Of this group, 34 (40%) were found incidentally at operation for other purposes or at autopsy (Table). This leaves a 60% incidence of symptomatic Meckel's diverticula, which is comparable to the figure reported by Söderlund.[4] Cases of Meckel's diverticulum which present pictures of clinical significance usually fall into one of four groups.

A. Ulceration.—With either gastric or pancreatic tissue in a diverticulum, the chance for peptic ulceration is present in a high percentage of cases. Waugh and his colleagues[5] demonstrated that such heterotopic gastric mucosa can produce the same secretions as a normal stomach. Other investigators[6] have demonstrated that this secretion can be stimulated by the same hormone that works to produce hydrochloric acid and pepsin in the stomach itself. Since there is no food present in the ileum to neutralize the presence of this acid, the chances for ulceration in this region are likewise greater than higher in the gastrointestinal tract.

Clinically, this ulcerative type is the commonest that one encounters in children who suffer from pathology of Meckel's diverticulum. This has been the experience of others and was likewise true in our series of 50 cases (Table), in which we had 23 patients who presented with this picture. This ulcerative type is manifest most commonly by painless rectal bleeding, varying in amount. It is usually changed blood that comes out of the anus because it has been acted upon by the gastric or pancreatic secretion of the diverticulum. If the hemorrhage is brisk, the anal evidence may be bright red. Of our 23 patients with peptic ulceration of the diverticulum, 17 presented with painless rectal bleeding as their chief complaint.

A few ulcers will have gone on to frank perforation, resulting in the classic picture of a child with marked peritoneal irritation and some degree of shock. The chief complaint in these cases is usually pain and/or vomiting, and this was true in the three cases of our series in which perforation had occurred.

Some writers report that this ulceration may be chronic and lead to stenosis of the ileum at the diverticulum as the ulceration heals. This results in intermittent intestinal obstruction or to an episode of complete obstruction. It has not been our experience to encounter this situation.

Still another manifestation of this ulcerative type may be what has been described as "dyspepsia Meckeli." This is seen in a child who has "feeding difficulties" and bouts of pain. Not infrequently he experiences pain 15 to 30 minutes after meals, when the irritative gastric secretion in the ileum is at its maximum, without any food to neutralize it. Usually the pain disappears within an hour after its onset, as the intestinal contents proceed down where they can vitiate the acidity. It is difficult to assess this manifestation accurately in reviewing a group of cases; children with chronic

abdominal pain who have exploratory laparotomy and removal of the Meckel's diverticulum may well be those who have either ulceration or simple inflammation of the diverticulum.

It is of interest to note that 21 of the 23 cases of the ulcerative group had gastric or pancreatic mucosa present in the pathologic specimen. In one of the remaining two cases the mucosa was read as normal. In the other, one of the perforation cases, it was felt that perhaps the islands of gastric tissue had been missed in the perforated area.

B. Inflammation.—Inflammation is not considered to be a common complication of Meckel's diverticulum because the diverticula are usually short with a wide base, which allows for free passage of the fecal stream in and out without the accompanying obstruction which may precede a true inflammation. Those diverticula which have a narrow base and a poor avenue of egress may well be liable to diverticulitis. The central abdominal location of the diverticulum lends seriousness to an inflammation that goes on to perforation, because generalized peritonitis will almost certainly result.

Inflammation of the diverticulum is very similar in its clinical picture to acute appendicitis, and almost all patients with an inflammatory type of reaction are operated upon with the preoperative diagnosis of acute appendicitis. Some people have pointed out that the localization of signs, beneath and around the umbilicus, may be a differentiating point. This does not seem to be universally true, and one would be hesitant to make a differential diagnosis on this one point. Brookes [3] has warned against failing to look for a Meckel's diverticulum in an acute abdominal condition in which only a slightly reddened appendix is found. He feels that this redness may well be due to peritonitis and that the true offending lesion —the diverticulum—will be overlooked unless a search is made for the Meckel's diverticulum in all cases of nonacute appendicitis.

In reviewing our own experience with the types of Meckel's manifestation, we were surprised to find that 14 of our 50 cases fell in this inflammatory group (Table). As mentioned above, there may be dispute as to whether some of these cases should be placed in the inflammatory or in the ulcerative group. Of the 14 cases, 10 had acute symptoms of peritoneal irritation, and yet only 3 of them had gastric mucosa in the resected specimen and melena as the chief complaint. If one wished to place these three in the ulcerative group, one could present a reasonable case for doing so. In three additional cases operation was performed because of inflammatory disease in the abdomen, but the resected Meckel's diverticulum merely showed chronic inflammatory disease and only one of the specimens presented aberrant gastric mucosa. The final case was one in which there was frank perforation in what appeared to be acute diverticulitis. Once again the resected specimen showed no aberrant mucosa, and this case was, therefore, placed in the inflammatory group.

C. Obstruction.—The third commonest clinical manifestation in our experience has been intestinal obstruction. The obstruction took one of the three common forms that are associated with this pathologic entity—intussusception, volvulus, or bands.

Intussusception is not an infrequent cause of difficulty in Meckel's diverticulum with obstruction. Abnormal peristalsis in that area produces invagination of the diverticulum as a leading point for the intussusception. The patient's clinical picture is that of one who vomits and has colicky pains, bleeding from the rectum, and, generally, a mass. This picture is usually sufficient for diagnosis; whether one utilizes a barium enema for further diagnosis and/or treatment depends upon one's convictions about this modality of diagnosis and/or therapy. Six of our cases had intussusception (Table).

An additional three cases had intestinal obstruction due to twisting of the intestine around a Meckel's diverticulum which was

attached to the underside of the umbilicus (Table). These all presented with vomiting and abdominal pain. The exact cause was not clearly delineated until exploratory laparotomy was undertaken for the obstruction. Occasionally one can gain a hint of the underlying pathology by rectal examination, actually feeling an attachment to the underside of the umbilicus.

Finally, it has been pointed out by a number of writers that the blood supply to a Meckel's diverticulum arises as a branch of the superior mesenteric artery and that, for all practical purposes, the diverticulum is supplied by an end-artery. This artery generally traverses the surface of the normal ileum and provides a potential hiatus through which an internal hernia may develop. There may be an abnormal attachment of the Meckel's diverticulum itself to some other viscus in the abdominal cavity, providing a hiatus for internal herniation. Finally, such an attachment may, itself, be so tight as to bring about an obstruction to the intestinal tract. Two of our cases of Meckel's diverticulum with intestinal obstruction were due to such bands (Table).

D. Umbilical Sinus.—Because of the embryonic development of this area, it is possible for a Meckel's diverticulum to give rise to symptoms solely in the area of the umbilicus. It may show itself in three general ways. First, the omphalomesenteric duct may be completely patent, in which case one may have a discharge of the fecal stream onto the abdominal wall by way of such a patent duct. The patient is brought to the physician because of the unpleasant odor and irritation around the umbilicus. The diagnosis can usually be arrived at by the ingestion of a nonoxidizable dye, which will appear in the discharge from the umbilicus. A variant of the patent omphalomesenteric duct is one in which the patency is so large as to allow an intussusception of the ileum through it. This anomaly has been encountered in two previous cases in my experience but is not included in the present series. Finally, the omphalomesenteric duct may become obliterated partially and leave only a very small umbilical sinus, which will manifest itself as a small knob of granulation tissue in the umbilical area with a thin discharge from it. The lesion from which one has to distinguish such a sinus is a patent urachus, and this can usually be done by injecting dye into the bladder and watching for its appearance at the umbilicus.

Only two of our series of cases presented as abnormalities of the umbilicus (Table), and both these diverticula were small remnants of the omphalomesenteric duct which drained clear fluid. Interestingly enough, both these specimens, when resected, had aberrant gastric mucosa present in them.

Treatment and Results

The question which universally comes up in discussing the treatment of Meckel's diverticulum is whether they should all be removed, or only those which are symptomatic.

Sixty per cent of the diverticula that have been brought to our attention have given rise to pathologic conditions; of the four clinical manifestations of this pathologic entity, all but the umbilical sinus are potentially lethal. It would seem wise, therefore, to adopt the policy that all Meckel's diverticula should be excised when seen, whether the finding be incidental or due to symptoms. This policy would, of course, be varied if the primary reason for exploratory laparotomy was so serious, and the child was in such precarious condition, that further surgery would endanger the child's life.

Two classic types of therapy have been applied to this condition. Simple diverticulectomy is done in those cases in which the diverticulum can be taken off without technically compromising the lumen of the bowel. It has been stated by most persons who have had experience in this field that such removal by diverticulectomy is completely effective because the presence of gastric mucosa is limited to the diverticulum itself and one may reasonably expect no

ectopic mucosa to be left after a diverticulectomy. This assumption has been borne out in our own experience, in which 30 of our patients were treated with diverticulectomy and a postoperative follow-up revealed that none of them had had any further trouble.

Intestinal resection should be reserved for those diverticula in which simple diverticulectomy would compromise the lumen of the bowel and lead to possible secondary intestinal obstruction. Resection with end-to-end anastomosis was utilized in 19 cases in our series.

These two forms of therapy resulted in an over-all mortality rate of 6% in the 50 cases seen, including the 1 patient who came to the hospital in a moribund condition and received no therapy. This experience compares favorably with the 6.6% reported between 1940 and 1950 from the Boston Children's Hospital [7] and the 8% and 21.6% reported in series from Sweden [4] and England,[3] respectively.

Summary

A series of 50 cases of Meckel's diverticulum seen at the Children's Hospital of Pittsburgh between 1940 and 1955 is surveyed briefly.

This lesion is discussed from the standpoint of its embryologic background and anatomic variations.

The four characteristic clinical pictures of this pathologic lesion are reviewed.

The types of therapy with their results are presented. There was a 6% mortality in this series.

125 De Sota St. (13).

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Inguinal Hernia in Infants and Children

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Inguinal hernia has harassed the physician for approximately twenty centuries. Its familiarity is attested to by its frequency in occurrence and its prominence in the medical literature. Its complexity is demonstrated by a persistence of different treatments since the Ebers Papyrus, in 1550 B. C.[1] Misinformation still exists concerning hernia of infants, probably because less attention has been brought to this young age group. There are some who still advocate trusses for the treatment of hernia in infancy and who advise waiting for surgery until the child "gets older." These recommendations are not without danger and probably should be replaced by a firmer stand that surgery be considered when the hernia is diagnosed.

All inguinal hernias of infants and children in my experience have been indirect. It is commonly agreed that the pathology of an indirect inguinal hernia is persistence of the processus vaginalis. Failure of obliteration results in a sac lined with peritoneum, which may extend from the internal inguinal ring to the testis. This peritoneum-lined cavity may be invaded by bowel, bladder, omentum, or any of the female genital organs. Whenever such a structure enters the sac, a visible lump or bulge appears. This may occur immediately after birth or any time later. Therefore, the sac is present at birth, but the external evidence of a hernia may appear at any later date. Since the sac is of congenital origin, it is unlikely that it will spontaneously cure itself. This does not mean that the inguinal bulge may not disappear and remain absent for some time. It seems reasonable that if the hernia is kept reduced constantly for a considerable length of time, the neck of the sac may narrow sufficiently no longer to allow anything to enter it. This assumption probably accounts for the temporary cures with the aid of a truss. The lump may be absent, but the sac persists, only to have some organ wedge its way into it at some later time. Since the etiology of an indirect inguinal hernia is a persistent patent processus, the logical cure would be to obliterate this sac.

Simplified treatment of inguinal hernia has been advocated by numerous writers in the past. Ferguson,[2] in 1899, suggested leaving the cord undisturbed during the repair. Herzfeld[3] recommended a repair that could be done as an outpatient procedure. Others have contributed various simplifications since. Potts,[4] in 1950, reemphasized the method advocated by Russel in 1925, in which the repair was confined to removal of the sac only. During the past 10 years at the Children's Memorial Hospital all hernias treated by the surgical department have been repaired by this method. Approximately 2000 hernias in infants and children have been operated on, the patients ranging in age from 17 days to 15 years. It is from a review of these cases and statistical data that the clinical impressions and recommendations described below have been formulated.

Statistical Data

Of 2110 hernias repaired at the Children's Memorial Hospital, 86.3% were in male and 13.7% in female children, or a ratio of 6:1. There were 1099 (54.4%) on the right side and 591 (28.1%) on the left, and 370 (17.5%) were bilateral. The youngest patient operated on was a 17-day-old infant who had an incarcerated hernia for 12 hours. The upper age limit was automatically confined to below 16 years because of the hospital rules.

Twenty-nine per cent of all the patients with hernias had a history of incarceration

sometime in the past or were admitted with the diagnosis of incarcerated hernia. Such a high percentage is difficult to evaluate because many with histories of incarceration were treated by the parent at home with ice bags, rest, or manual reduction. However, 18% of the total number with hernias were admitted with a diagnosis of incarcerated hernia which required sedation for reduction or were irreducible. Only 65 (3.2%) cases of incarceration did not respond to conservative management and needed immediate surgical treatment. Most of the incarcerations can be reduced manually if done properly.

Diagnosis

The diagnosis of a large hernia is usually made by the mother. The bulge or lump in the groin is noticed, and it may even protrude into the scrotal sac. Such findings present no diagnostic problem. All hernias are not so evident. Some are discovered because the child complains of pain in the lower abdomen. Infants may show only signs of irritability or fussiness. In these patients a light, but deft, index finger can usually distinguish the normal spermatic cord from one that contains a small hernia. It is impossible to insert a finger into the inguinal canal of an infant, and the older children rebel so much that it usually is not feasible to attempt such a maneuver. A simple, and yet accurate, way to palpate a hernia is to place the index finger lightly over the inguinal canal just below the level of the internal ring (Fig. 1). By gently moving the finger from side to side, rolling the cord beneath it, a fine "swish" can be felt if a hernia is present. This sensation is sometimes referred to as the "silk glove" feel and merely represents the two surfaces of peritoneum rubbing together. It is often easier to feel this "swishing" sensation if more fluid is forced into the hernia sac by increasing the intraperitoneal pressure. This can be done by pressing on the abdomen of the child or by making the infant cry. By repeatedly feeling the "swish" and finding a contrast to the opposite side, a diagnosis of inguinal hernia usually can be made. Early recognition of a hernia may solve the problem of an unusually fretful baby. Each time the infant cries or strains the hernia protrudes and is painful.

Not infrequently an infant is brought to the physician because the mother discovers a hard "seed" or nodule in the inguinal region. It commonly is not tender unless it has been palpated too vigorously. Such a nodule is located immediately beneath the internal ring (Fig. 2). It is quite a common

Fig. 1.—Technique of palpating for an inguinal hernia in a child.

Fig. 2.—Incarcerated ovary in a right inguinal hernia.

finding and, when present in a female child, is probably an ovary. Often it is irreducible, but seldom does it need immediate repair. Only if repeated energetic reduction is attempted does the ovary become swollen and tender. Usually the ovary can remain in the hernial sac, even though it cannot be reduced, without impairment of its blood supply, and surgery can be done electively. If without manipulation the ovary (nodule) appears to be tender, it should be treated as an incarcerated hernia, and surgical correction be done immediately.

If this type of isolated nodule is felt in the inguinal canal of a male child, it could represent a hydrocele of the cord but should be evaluated carefully. The differential diagnosis of a tender lump in the inguinal region of an infant is truly challenging. Certainly, inguinal abscess, hydrocele of the cord, and incarcerated hernia must be considered each time one is confronted with inguinal tenderness. The history is most unreliable and usually helps very little. It may be impossible to differentiate an incarcerated hernia from a hydrocele of the cord. Usually the hydrocele has more definite margins, so that the cord can be felt above the mass as well as below, in contrast to an incarcerated hernia. Hydroceles are not tender, whereas incarcerations frequently are. If there is any doubt as to the diagnosis, an operation should be performed. It may be slightly embarrassing, to operate upon a hydrocele of the cord as an emergency, but much more expedient than to observe an incarcerated hernia progress to strangulation. Inguinal abscess is not uncommon and must be distinguished from an incarcerated hernia. Very seldom is the skin red or indurated with an incarcerated hernia, whereas skin involvement is usually present with an abscess. The inguinal lymph nodes, which are the nodes commonly infected prior to an inguinal abscess, are located lower and more laterally than the inguinal canal. One should not be misled by scrotal edema. This can be present with an incarcerated inguinal hernia, as well as an inguinal abscess. Scrotal abscesses in infants and children are very rare and should be diagnosed cautiously.

If the diagnosis of an inguinal abscess is made and incision and drainage considered, the abscess should be exposed layer by layer rather than by a stab type of incision. If the diagnosis is questionable, an x-ray may be helpful in demonstrating the gas pattern of a loop of bowel in the scrotum (Fig. 3). The infant shown in this Figure was brought to the hospital with small-bowel contents draining from a scrotal incision. The patient had an incarcerated hernia, which was thought to be an inguinal abscess. Two abdominal operations were necessary to correct the fecal fistula. After three months of hospitalization, the child was finally discharged—all because of an inguinal hernia!

Incarceration

The importance of a hernia immediately changes when it no longer is asymptomatic and freely reducible. Incarceration is feared by parent and physician alike, and rightfully so. The omentum is underdeveloped in children and rarely becomes incarcerated. The structure involved is almost always

Fig. 3.—Upright film of an incarcerated inguinal hernia giving bowel obstruction.

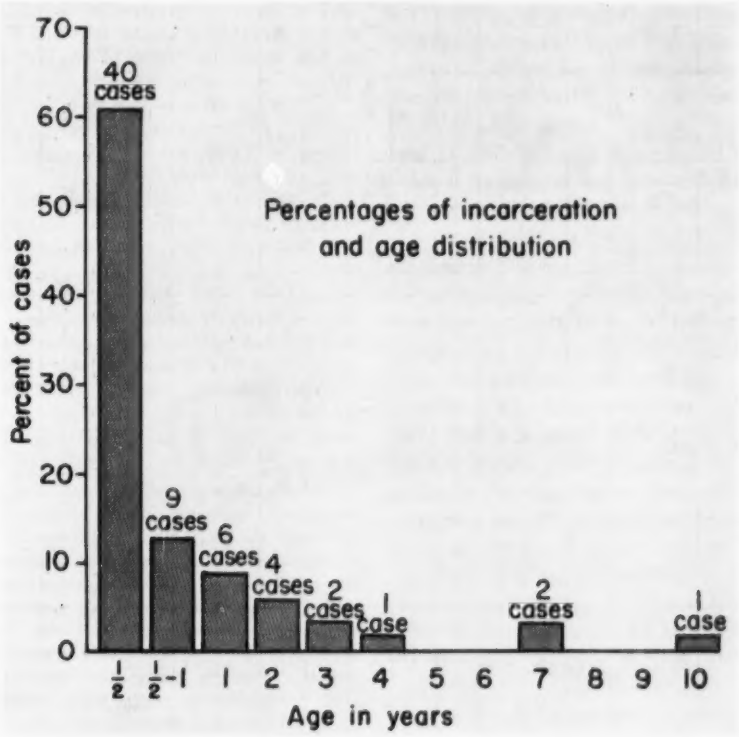

Figure 4

bowel in the male child and either bowel or ovary in the female.

It is interesting to note that the highest incidence of incarceration is found in the young age group. In 65 cases of incarcerated hernia operation was performed because conservative attempts at reduction failed. Figure 4 reveals the age distribution of children with incarcerated hernias that needed operative reduction.

Symptoms of incarceration vary from restlessness to severe distention and vomiting, such as are seen in mechanical bowel obstruction. The visible mass in the inguinal canal or scrotum usually is tender. It is difficult to reduce a hernia that is tender because the moment pressure is applied the infant cries so forcibly that all attempts to push the hernia back are met with failure. Because the tissues are edematous and repair is more difficult during incarceration, a trial of conservative treatment is worth while.

Fig. 5.—Child in modified Bryant's traction to reduce an incarcerated inguinal hernia.

This conservatism consists of placing the child in a modified Bryant's traction and giving him phenobarbital sodium by injection for sedation (Fig. 5). Often the hernia will reduce itself by this simple technique. If reduction is accomplished, the child is kept in the hospital and the operation is done after about 48 hours. By this time the edema has disappeared and the operation is performed with greater ease and safety. If the hernia is not reduced within one to two hours after conservative therapy has been instituted, the child is prepared for surgery.

Of 65 children whose incarcerations were operated on, only two required resection of bowel. Both these children did well postoperatively. However, there were five cases in which the testes were black, one of which was removed because of obvious gangrene. If at all possible, the testis is left in place, in the hope that at least some of its hormone activity will remain. None of the dark-looking testes left in place caused any difficulty later. They became atrophic but did not slough or form an abscess.

In the female children all the incarcerations involved the ovary except one—a 10-year-old girl who had a portion of small bowel incarcerated. All incarcerated hernias of female infants contained only the ovary. Only one ovary showed any sign of vascular insufficiency, and it resumed its normal color soon after being freed.

Operative Technique

When the diagnosis of an inguinal hernia has been made, repair is advised. Surgery is recommended regardless of the age of the child, provided the baby is well and gaining weight. Postponing repair invites complications and prolongs anxiety for the parents. If the hernia is repaired when first noticed, perhaps the high incidence of incarceration in infants can be reduced. Most physicians are aware that an uncomplicated hernia can be repaired safely, easily, and permanently by a simple operation. Why wait until the operation becomes an emergency, fraught with more danger and difficulties?

Ordinarily, in a case of uncomplicated hernia the child is admitted to the hospital the day before surgery. This enables one to obtain a careful examination, a blood count, a urinalysis, and a brief period of observation in order to be sure there is no evidence of any infectious disease. At no time should the hernia be repaired if there is any sign of an upper respiratory infection.

The child is given nothing to eat or drink after midnight, and the following morning the operation is done, with general anesthesia. Open-drop ether anesthesia is routinely used.

The skin of the genitalia, groin, and lower abdomen is prepared with ether, iodine, and alcohol, a method a bit old-fashioned but still effective.

The incision is made parallel to the normal skin folds of the lower abdomen. This incision lends itself to very easy closure, with minimal scar, and is easily bandaged to avoid contamination postoperatively. The oblique incision is difficult to bandage adequately, especially in infants, who so quickly soil the dressing. The incision is made just below the level of the internal ring. In infants this usually lies in the suprapubic fold (Fig. 6).

Scarpa's fascia is a very definite layer, especially in infants, and must not be mistaken for the fascia of the external oblique muscle. The external oblique is cut parallel with its fibers toward the external ring. If the external ring is not opened, there is no problem in reapproximating it with a proper-sized opening. The cremasteric fibers over the cord are gently separated with a hemostat, and the hernia sac can be seen immediately beneath as a gray-white structure. The sac is grasped and gently dissected free from the structures of the cord. Usually, the vas deferens is the structure most adherent to the sac. The sac is dissected free as it is elevated, leaving the vessels and vas undisturbed if possible. By meticulous sharp dissection the sac is mobilized from the structures of the cord. Properitoneal fat is exposed and freed from the neck of the sac. The sac is opened, reduced of its contents, and twisted to change it from an extremely fragile, thin-walled, easily torn structure, to a stronger, twisted cord. This permits one to pull on the sac to obtain a higher ligation without danger of tearing. High suture ligation of the sac is the curative step of this operation and must be done exactly and carefully. After the sac has been ligated high, the important prerequisite for cure, the excess is amputated, and the stump promptly retracts beneath the internal oblique muscle.

The wound is then closed in its normal layers. The external oblique fascia is sutured with interrupted fine nonabsorbable surgical (silk) sutures. The edges of Scarpa's fascia are reapproximated, and the skin is closed with a couple of subcuticular stitches of 000000 silk in infants below 18 months, or with a continuous through-and-through skin suture of 0000 silk in older children. The wound is covered with a small strip of gauze and waterproof adhesive tape.

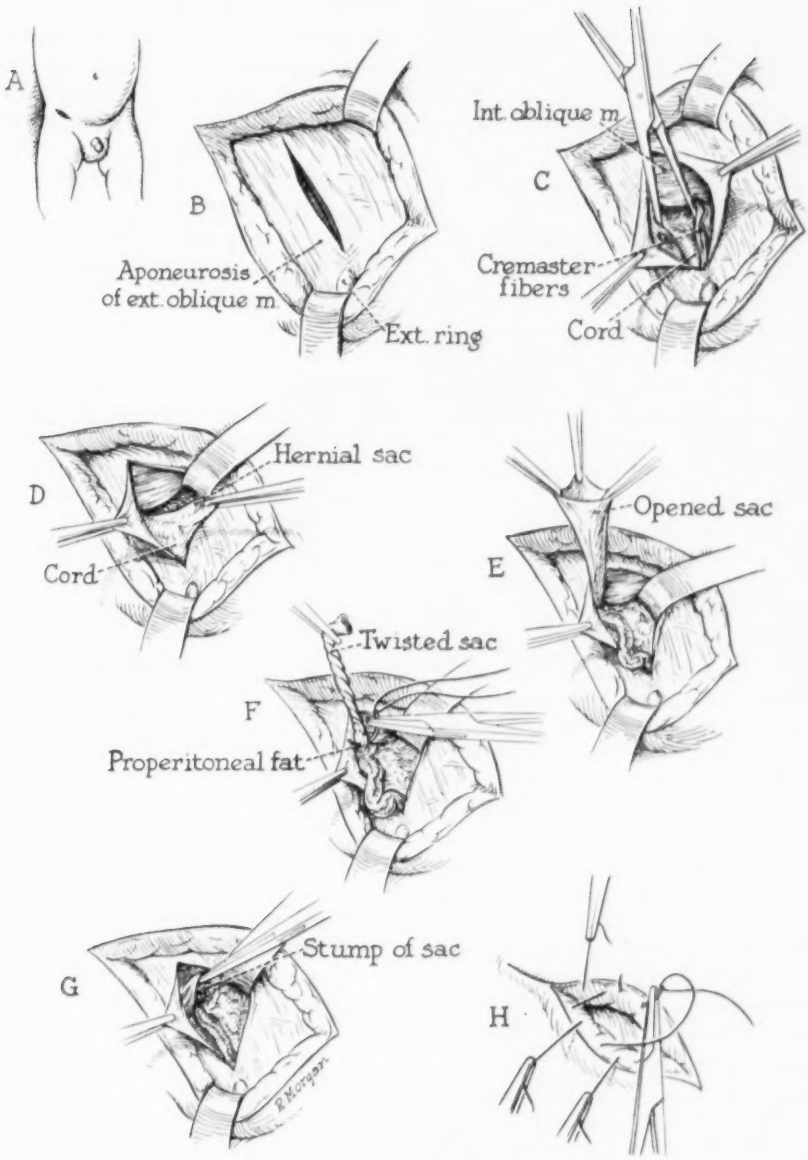

Fig. 6.—Drawings of the steps in repair of an inguinal hernia.

The postoperative care is usually simple. Infants of less than 18 months are fed approximately six hours after surgery and seldom vomit, even though general ether anesthesia is given. The older children often undergo the usual discomforts of nausea and vomiting until late that evening. The following day the patient is discharged and is allowed relatively free activity, except the older children, who are restricted to less strenuous play.

Postoperative complications are minimal. An occasional minor wound infection, and some swelling of the testis, especially if there was an associated hydrocele, are about the only noticeable features. Hematoma within the cord or around the

testis is avoided if special care is taken while dissecting the sac free and if complete hemostasis is accomplished, especially of the small blood vessels of the cord. Testicular edema and pain resulting from closing the external ring too tightly are avoided in this type of repair because the ring is not opened.

Results

The parents are uniformly grateful after the operation. They have credited the operation with being a cure for many symptoms. It is difficult to evaluate these benefits because they are only the interpretations of a parent. At least the parents no longer have to worry about the reducibility of the hernia, struggle with reapplying a cumbersome truss, or be concerned whenever the baby cries.

The recurrence rate is always a consideration in a surgical procedure for hernia repair. In the entire group of 2110 hernias, there are 7 known recurrences. Of the seven known recurrences, there were three in female and four in male children.

Because of the surprisingly high recurrence rate in female children, an attempt was made to find the reason for the recurrence. It was found in one case, of incarcerated hernia in an infant less than 4 months of age, that the probable cause of the recurrence was a technical error, such as making a small tear in the peritoneum while suturing the sac. A small tear is difficult to recognize when all of the surrounding tissue is edematous from the incarceration. Two recurrences were in patients who had a sliding type of hernia, the tube and ovary being the lateral wall of the sac.

Sliding hernias are common, especially in female children. A review of 173 hernias in girls revealed 44 (25.4%) of the sliding type. Some involved only the tube; others included tube, ovary, and fundus of the uterus. The routine type of suture ligation of the sac cannot be done in these cases because of the possibility of occluding the blood supply to the involved ovary. This problem instigated a modification in the repair of a sliding hernia in female infants. Instead of closing the neck of the sac in continuity, a tongue of peritoneum which contains the broad ligament and ovarian artery is cut, isolating it from the remainder of the hernial sac. This tongue of peritoneum is then replaced in the peritoneal cavity, and the sac is closed with a purse-string type of closure.

Of the four recurrences in male children, one was in an infant with gargoylism who had a huge bilateral hernia. Children with this abnormality, who seem to have a lack of adequate structural material, need a more complete repair to strengthen the inguinal floor than simple ligation of the sac. This also holds true in infants with extrophy of the bladder. A reconstruction of the inguinal floor is advocated in these types of patients. There were three recurrences in male children that probably represented a technical error. Two of these children had recurrences of a sac, and the previously placed silk suture was incorporated in the sac structure. Repair was done a second time by the high-ligation method, and the children have had no further trouble. The third child was taken to another surgeon for the second repair. All the recurrences were noticed within one year of the repair.

A follow-up study of 100 cases of hernia repair was carried out, extending over a postoperative period of three to five years. In this group there were no recurrences and no testicular atrophy. Five developed hernias on the opposite side—three on the left after repair on the right side and two on the right after repair on the left side.

One postoperative death in the entire group of 2110 hernias was that of a premature infant who was admitted to the hospital moribund with a strangulated hernia. This infant weighed 4 lb. (1814 gm.) and had been ill four days. The hernia was operated upon, but the child died postoperatively.

An occasional superficial wound infection occurred, which subsided spontaneously. Some infants seemed to tolerate the silk sutures poorly, and the wound failed to

heal properly until the silk finally worked its way out or was removed. At no time has it been necessary surgically to remove the silk sutures that were used below the subcutaneous layer.

Summary

During the past 10 years 2110 hernias were repaired at the Children's Memorial Hospital, using simple high ligation of the sac, as described. There have been seven known recurrences to date. There was one postoperative death.

Sixty-one per cent of the incarcerated hernias occurred in infants under 6 months of age. Repair of the hernia is advised as soon as the diagnosis is made. All incarcerated hernias in female infants contained ovary and usually do not require immediate surgery. Incarcerated hernias in male children almost always contained bowel and must be reduced as soon as feasible. Since most of the incarcerations can be reduced, conservative therapy is suggested as the preliminary treatment.

Almost one-fourth of the hernias in female children are of the sliding variety and need a slightly modified repair to prevent recurrence.

A tender inguinal mass in a child may be an inguinal hernia, hydrocele of the cord, or an abscess and must be differentiated.

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Cardiopulmonary By-Pass in Surgical Treatment of Congenital or Acquired Cardiac Disease

Use in Three Hundred Five Patients

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The past decade has brought forth an impressive display of clinical achievements in the field of cardiac surgery. As recently as 1948 there were only three congenital cardiac lesions then amenable to surgical treatment. These were patent ductus arteriosus, coarctation of the aorta, and the systemic-pulmonary artery anastomotic procedure for tetralogy of Fallot. In effect, the interior of the heart represented an important barrier to progress, the last anatomic frontier of the many that have confronted surgeons through the generations.

The present important advance in cardiac surgery has been the development of the open operation, in which the surgeon sees precisely what has to be done within the cardiac interior chamber and then proceeds to carry out the necessary reparative measures in a dry field, unhurriedly.

Every advance such as this brings the possibility of a fuller life to many who are at present disabled or handicapped. The magnitude of these developments can be judged from a consideration of the numbers of patients with congenital and acquired heart disease in the community. At present some 50,000 infants are born with congenital heart disease in the United States each year. In the world as a whole, at present birth rates, 2000 infants arrive every 24 hours with congenital defects of the heart. Most of these infants are potential candidates for corrective surgery. Probably three-fourths, or more, have curable lesions utilizing these currently available open methods, and all but a few of the remaining infants can be assisted through surgery. The estimation of those numbers requiring relief from acquired heart disease is admittedly less accurate. A conservative prediction would be that at least 25,000 persons yearly in this country could be benefited utilizing only the present possibilities of surgical treatment.

These large numbers of patients emphasize the need for widely acceptable and widely applicable methods for detection and treatment of cardiac diseases. Lest there be any complacency, one need only reflect upon the inescapable fact that more children with intracardiac defects are born each day than are being corrected in a year with present existing facilities. This backlog of undiagnosed and untreated patients remains less alarming only because of the high attrition rates for many of these malformations in the first years of life, as well as later on.

Therefore, it is quite predictable that the practice of cardiac surgery, particularly open cardiac surgery, will not remain confined to a few medical centers but will continue to grow, of necessity, at an impressive rate.

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Development of Open-Heart Surgery

The experimental work of Gibbon,[1] beginning in 1939, focused interest upon extracorporeal oxygenating techniques. A clinical application of his type of pump-oxygenator, as modified by Dennis,[2] was attempted at the University of Minnesota in 1951. Both operations were for an atrial septal defect, and were unsuccessful. In 1952 Lewis[3] and colleagues successfully, for the first time, repaired under direct vision an atrial septal defect (secundum type). In this young girl general hypothermia was utilized after the method suggested by Bigelow.[4] Closure of atrial septal defects under direct vision utilizing hypothermia thereafter became a regularly scheduled procedure. However, despite successes with closure of the secundum atrial septal defects, the limitations of time and myocardial irritability for intracardiac work under hypothermia quickly became evident. Attempts made to apply this method to the atrioventricularis communis defects or ventricular septal lesions proved disappointing.

In turn, Gibbon,[5] in 1953, reported the first successful repair of an atrial (secundum) defect under direct vision utilizing total cardiopulmonary by-pass with the aid of his pump-oxygenator. Additional good results with either this type of lesion or with ventricular septal defects remained difficult to achieve.

During this period, while the use of hypothermia became commoner and interest in it grew strong at several institutions, including the University of Minnesota, research here continued equally vigorously on the development of methods permitting intracardiac surgery at normal body temperature. These efforts were fostered by the growing recognition, from many hours spent in the autopsy room dissecting and suturing fresh specimens of congenital heart disease, that a longer interval than that available under hypothermia would surely be required for the precise repair of the more complex atrial and all ventricular septal defects. Too, the group was acquiring laboratory[6] and a limited clinical experience[7] with the possibilities under inflow stasis. This information, bolstered by the azygos flow[8] ultimately led to a concept of cardiac and systemic tolerance for much lower perfusion

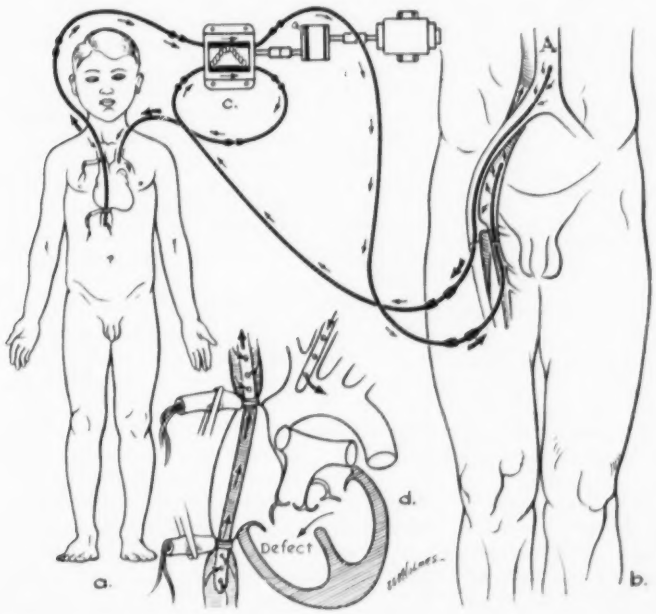

Fig. 1.—Method for direct-vision intracardiac surgery by means of controlled cross circulation: (*a*) Patient, showing sites of arterial and venous cannulizations; (*b*) donor, showing sites of arterial (superficial femoral) and venous (great saphenous) cannulizations; (*c*) the pump which controls the reciprocal exchange of blood between patient and donor; (*d*) close-up of the patient's heart, showing the vena caval catheter positioned so as to draw venous blood from both the superior and the inferior vena cava during the cardiac by-pass interval. The arterial blood from the donor is circulated to the patient's body via the catheter inserted into the right subclavian artery.

TABLE 1.—*Intracardiac Correction of Congenital Anomalies by Controlled Cross Circulation*

Results in Patient's Defect	Direct-Vision Operative Procedures	No. of Patients	Results	
			Successful	Deaths
Ventricular septal defect	Suture closure of defect	27	19*	8†
Tetralogy of Fallot	Closure of ventricular defect and correction of pulmonary stenosis	10	5	5
Atrioventricularis communis	Closure of ostium primum with suture repair of atrioventricular valvular deformities	6	1‡	5
Isolated infundibular pulmonary stenosis	Resection of infundibular muscle	1	1§	0
Pulmonary stenosis, with atrial septal defect and anomalous pulmonary venous drainage into superior vena cava and right atrium	Pulmonary valvulotomy, closure of atrial defect, and transposition of pulmonary veins to left atrium	1#	0	1
Total		45	26	19

* Postoperative recatheterization was done in 16 patients, and in 12 there is no shunt. One of these 12 had both atrial and ventricular cardiotomies for coexisting separate defects. Four patients have a residual shunt, and in all four the pulmonary pressures have fallen. Three of these four have normal pulmonary pressures postoperatively, indicating that residual shunt is small.

† Six of the deaths were in infants under the age of 2 years.

‡ This patient has been recatheterized and no shunt remains (Table 7).

§ The patient has been recatheterized and the preoperative pulmonary artery pressure, of 136/0 mm. Hg, was (four months postoperatively) 25/10 mm. Hg.

An 11-month-old infant.

Donor results:

No. of Donors.... 45 Early-Late Mortality.....0

rates than other workers had considered possible.

Methods for Open-Heart Surgery

Controlled Cross Circulation.—To eliminate some of the already mentioned shortcomings of the then existing methods, use of a donor circulation was conceived (Fig. 1). This circuit would oxygenate and revitalize the patient's blood. An external pump was added to control accurately the interchange of blood, which in volume was reduced substantially, on the basis of the azygos-flow experimental studies. This method of controlled cross circulation was believed likely to fulfill the objectives deemed necessary to ensure the successful intracardiac repair of the more complex congenital lesions. In its experimental trials it had proved wholly effective. The surgeon had available a prolonged time for working within the open heart and in a relatively blood-free field. After extensive testing these principles and methods in the research laboratory, the plan was applied clinically on March 26, 1954, for the successful repair of a ventricular septal defect.

Forty-five patients were operated upon during 1954 and early 1955 by this method. These results are summarized in Table 1. Its use was confined entirely to patients with complicated defects. Most of the patients suffered from advanced degrees of cardiovascular impairment. This technique made feasible, for the first time, completely corrective surgery for these lesions: ventricular septal defects, atrioventricularis communis, and tetralogy of Fallot. All persons previously afflicted with these anomalies were either incurable or subjects for palliative procedures only. More important perhaps than these initial patients fortunate enough to achieve objective cures for complex intracardiac defects was the fact that this clinical experience with cross circulation clearly indicated, contrary to what many had contended, that extensive reparative surgery within the hearts of even seriously ill patients was both possible and lifesaving. As a matter of fact, the technique of controlled cross circulation proved to be physiologically superb for those seriously ill patients. Once the circulatory systems of the patient and the donor were linked, the invalid was assured a continuous supply of normally oxygenated, warmed blood from the healthy donor, and also this mutual circulation was constantly undergoing appropriate adjustments through the numerous homeostatic mechanisms of the donor's lungs, kidneys, liver, and other organs. Both in theory and in practice, it is unlikely that a technique for total cardiopulmonary by-pass will be developed which, for the patient's safety, possesses more advantages than this one.

Meanwhile, stimulated by the demonstrated fact that these more complex intracardiac defects could be corrected, work pushed ahead rapidly on the development of nondonor blood oxygenators. By 1955, the Mayo Clinic group,[9] through their modification of the Gibbon machine, were able to report clinical successes after intracardiac operations. Meanwhile, during 1954 and 1955, from the University of Minnesota cardiovascular research group came three nondonor methods: perfusion from a reservoir of arterialized blood,[10] use of a specially prepared biological (dog lung) oxygenator,[11] and a disposable plastic bubble-diffusion

TABLE 2.—*Methods of Extracorporeal Oxygenation Utilized in Conjunction with a Mechanical Pump for Open Cardiotomy*
University of Minnesota Hospitals

Method of Oxygenation	Era Used	No. of Patients
Controlled cross circulation	1954–1955	45
Perfusion from a reservoir of arterialized venous* blood	1955	5
Biological dog-lung oxygenator	1955	15
Artificial oxygenator	1955 to present	240
Total		305

* Drawn from the veins of donors' previously warmed arms.

oxygenator.[11] The first, while practicable for infants, provided for larger patients too brief a duration of by-pass with any reasonable-sized reservoir. The second, while successful in its clinical oxygenation of human blood, would unpredictably result in pulmonary edema to the extent that its usefulness was limited. Since early 1955, the method of choice in this clinic for patients requiring open cardiac procedures has been the artificial oxygenator of a simple, disposable, bubble-diffusion type. The same type of pump utilized initially with controlled cross circulation has continued to prove satisfactory. The number of patients treated utilizing each of these oxygenating methods is summarized in Table 2.

Cardiac Lesions Managed by Open Cardiotomy.

The availability of effective methods for working within the open heart has permitted the application of visual repair to an ever-increasing variety and number of congenital and acquired malformations of the heart.

The present report, based upon 305 such operations, reviews the status of open-heart surgery at the University of Minnesota Heart Hospitals. Those lesions, so managed, are summarized in Table 3.

Cardiopulmonary By-Pass—Present Technique.

The standard technique for total cardiopulmonary by-pass is diagrammed in Figure 2. The patient's venous blood is allowed to flow by gravity from the caval cannulae into a plastic reservoir (not shown in Figure) and then is pumped into the oxygenator. The venous blood rises on the oxygen column through the oxygenator, and, after debubbling occurs, it is moved by gravity and, after being arterialized, is returned through filters by the arterial pump to the patient's arterial system. All of the catheters, the tubing,* and the oxygenator itself are of polyvinyl plastic. They are steam sterilized in an autoclave and are dis-

* The tubing actively compressed by the pumping Sigmamotor fingers is of pure gum rubber.

TABLE 3.—*Data on 305 Patients Undergoing Open-Heart Surgery During Cardiopulmonary By-Pass with Total Body Perfusion*
1954-1957
University of Minnesota Hospitals

Lesion	No. Cases Treated	Survival *	Per Cent Mortality
Ventricular septal defect	154	109	29
Tetralogy of Fallot			
Cyanotic			
First operation	60	40	33
Previous shunt procedures	5	0	100
Acyanotic	6	5	16.6
Atrioventricularis communis	20	11	45
Atrial septal defect (secundum)	12	12	0
Plus valvular pulmonary stenosis	1	1	
Plus pulmonary stenosis (trilogy), cyanotic	3	3	
Pulmonary stenosis, anomalous pulmonary venous connection	1	0	
Isolated pulmonary stenosis			
Valvular (acyanotic)	3	2	
Infundibular (acyanotic)	1	1	
Atresia (cyanotic, infants)	5	0	
Total anomalous pulmonary venous connection			
Superior vena cava	1	0	
Coronary sinus	2	1	
Ruptured aneurysm of sinus of Valsalva	3	3	
Aortic-pulmonary window	1	0	
Complete transposition of great vessels	5	0	
Aortic stenosis			
Congenital			
Valvular	1	1	
Infundibular	2	1	
Acquired, calcific	5	3	
Mitral Stenosis			
Congenital	3	3	
Rheumatic	3	2	
Mitral regurgitation—rheumatic	2	2	
Aortic regurgitation—rheumatic	2	1	
Exploratory ventriculotomy (patent ductus)	1	1	
Exploratory ventriculotomy (primary pulmonary hypertension)	1	1	
Aneurysm of aortic arch (resection and grafting)	2	1	
Total	305	204 (67%)	

* Operative and late deaths are included.

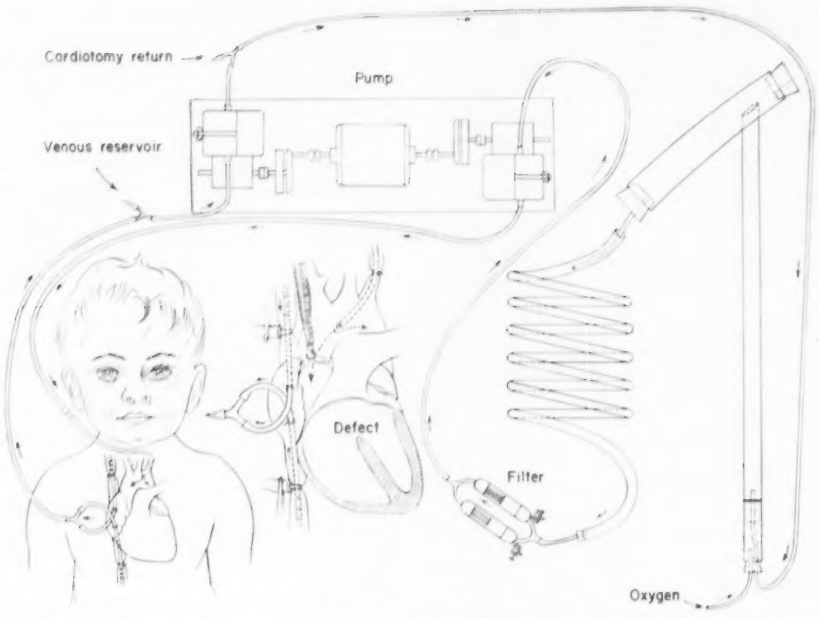

Fig. 2.—Diagram of extracorporeal circulation during total cardiopulmonary by-pass utilizing the helix-reservoir pump-oxygenator.

carded after a single use. The connectors and adaptors are polished stainless steel and are reused after appropriate cleansing and sterilization.

During the perfusion interval blood aspirated from the interior of the heart and operative field is returned directly to the oxygenator by the low-pressure cardiotomy sucker system.

For congenital malformations, such as ventricular septal defects, tetralogy of Fallot, and pulmonary stenosis, surgical exposure is by means of an anterior bilateral transverse thoracotomy in the fourth interspace. The sternum is transected in the midline. Before dividing the sternum, the internal mammary arteries are exposed and divided, and one of these can then be proximally cannulated with a small polyethylene catheter and connected to a recording device to provide conveniently a continuous record of arterial pressure. Upon opening the pericardium, the superior mediastinum is inspected for a left superior vena cava. In all patients with pulmonary hypertension due to congenital heart disease the ductus arteriosus area is dissected out routinely to determine patency. Both these congenital anomalies are frequent associated defects (Table 3) and interfere with perfusion if they remain undetected. For acquired heart disease of the aortic and mitral valves and such congenital defects as atrial septal defects, atrioventricularis communis, and certain forms of total anomalous pulmonary venous connection, the surgical exposure is often obtained through a right thoracotomy with the patient in the lateral position. Specially designed, thin-walled plastic catheters are used for the vascular cannulations. The outflow from the superior and inferior venae cavae is obtained by transauricular by-positioned catheters. To ensure the completeness of this return during cardiac by-pass, the cotton tapes, which have previously been placed around the cavae and passed through a short length of rubber tubing, are tightened around these caval cannulae by cross clamping this rubber sleeve (Fig. 2). A plastic catheter into either of the subclavian arteries or the femoral artery serves for return of the arterialized blood. At the end of the by-pass procedure the subclavian artery is ligated in infants and smaller children and repaired in adults. A femoral arteriotomy is always repaired.

Just before insertion of these great-vessel catheters the patient is heparinized (1.5 to 2.5 mg/kg. of body weight), and after their removal protamine sulfate (3 to 5 mg/kg. of body weight) is given to counteract the heparin. In these heparinized patients hemostasis must be more meticulous. With rigid attention to this detail, hemorrhage has not been a problem except in unusual circumstances.

The pump-oxygenator is primed with approximately 2 pt. (1000 cc.) of heparinized blood drawn within 24 hours of use. Heparinized blood is used for any replacement needed during the actual perfu-

sion. Before and after this interval citrated blood is used.

The patient's body temperature is monitored by a recording rectal catheter. Specific efforts are made to keep body temperature at normal levels during the entire operation so as to avoid the depressing effects of hypothermia upon the myocardium or the increased oxygen demands of hyperthermia. This temperature control is effected by warming the blood in the arterial reservoir of the oxygenator to 41 C and by placing a mattress under the patient through which either heated or cooled fluid may be circulated.

During operation all patients are monitored by a portable electroencephalographic recorder.

Bubble-Diffusion Pump-Oxygenator.—For intracardiac surgery we have had our greatest experience with the bubble-diffusion oxygenator. This has proved a simple, yet efficient, method, in which venous blood is filmed directly upon the surface of large bubbles of the oxygenating mixture (97% oxygen, 3% carbon dioxide). This process creates a large blood-oxygen surface area, which is in intimate contact for the rapid interchange of oxygen and carbon dioxide as the blood gently rises in the mixing tube. Foam formation at that time is undesirable and is evidence of improper technique. Next, arterialized blood spills into the silicone-coated plastic walls of the debubbling chamber. Simultaneously, the excess carbon dioxide and oxygen escape, and the arterialized blood descends by gravity flow into the helix reservoir. Figure 3 portrays this apparatus, which has been utilized in the majority of the patients here reported.

Unitized Disposable Sheet Oxygenator.—Because of both its simplicity and its satisfactory performance, the helix-reservoir bubble oxygenator was adapted in principle to produce a model suited to commercial production in quantity. The attainment

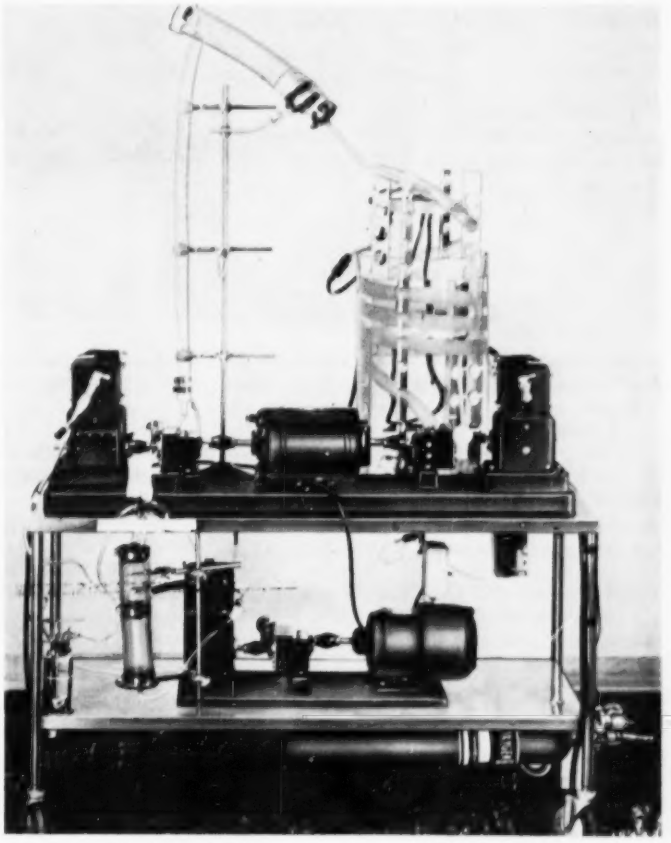

Fig. 3.—The pump-oxygenator utilized for direct-vision intracardiac surgery.

The pump consists of a single, explosion-proof electric motor, which activates two pump heads (arterial-venous), each equipped with an individual speed changer, all mounted on a single base. The artificial (large-bubble) oxygenator has no moving parts, is sterilized by autoclaving, and is constructed entirely of polyvinyl plastic tubing, which is discarded after each perfusion. From right to left may be noted the vertical mixing tube, the transverse debubbling chamber, and the helix reservoir, immersed in a constant-temperature water bath. From the lower end of the helix the oxygenated blood is returned through disposable filters to the patient's arterial system. The oxygen gas enters the venous blood directly through a nylon diffusion plate at the base of the mixing tube. The venous-blood carbon dioxide escapes with the excess oxygen during oxygenation.

The coronary-sinus return pump and reservoir for returning to the oxygenator blood aspirated from the open heart are seen on the lower level.

This pump-oxygenator has been utilized now in more than 500 patients in 14 different medical centers.

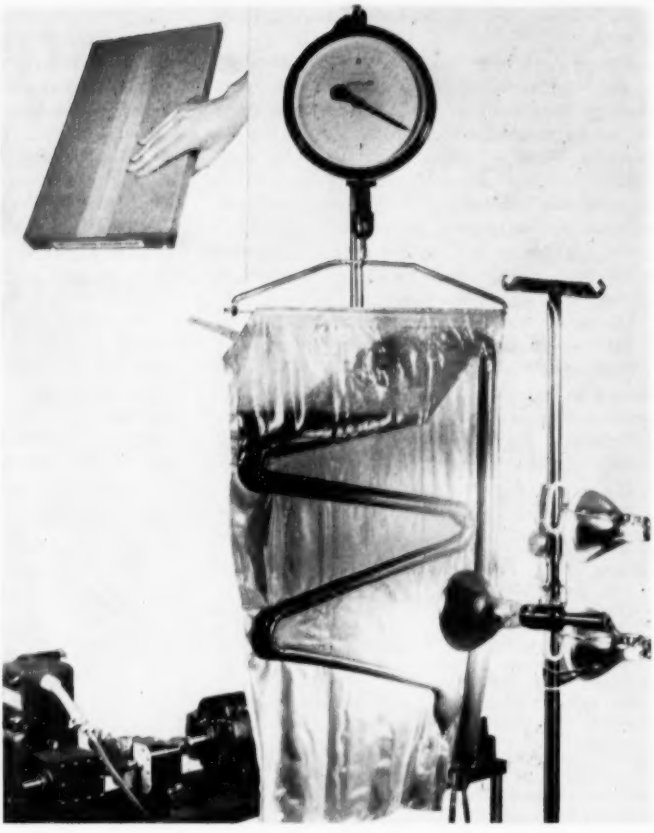

Fig. 4.—Photograph of unitized, disposable bubble oxygenator of plastic sheet in combination with the Sigmamotor pump during perfusion. Note the suspending spring scale, which allows instantaneous assessment of the blood volume. Heat loss is being controlled by the three heat lamps directed on the settling chamber. These lights are automatically turned on and off as necessary by the simple thermostatic relay switch inserted in the special pocket.

Inset: This oxygenator is available as a sterile packaged unit, ready to hang up, prime, use, and then discard.

of this objective not only should facilitate the development of open cardiac surgery in those centers where it is already being used, but also should assist in the growth of this technique in many other areas with less experience, solving the problems inherent in this type of surgery. This unitized oxygenator is constructed from two sheets of polyvinyl plastic. The desired channels and chambers are delineated by a heat seal (Fig. 4). The same chambers are present as in the three-dimensional prototype: vertical mixing tube, a Siliconized debubbling chamber, and a series of inclined settling columns. The oxygen disperser is heat-sealed into the lower end of the mixing chamber, and a saran (thermoplastic resin) filter has been fabricated into the arterial exit from the settling chamber. Two venous inflow tubes enter the lower end of the mixing tube. One tube accepts venous inlet to the oxygenator from the venae cavae, and the other carries the return flow from the cardiotomy aspirator.

Elective Cardiac Asystole.—The use of cardioplegic agents to stop temporarily the beat of the heart with concomitant clamping of coronary arterial flow allows full realization of one of the major goals of open-heart surgery, namely, an operative field that is both dry and motionless. We have used potassium citrate for this purpose in 43 patients and acetylcholine in 2 patients of this series.

This remedy has proved most useful in those septal defects where exposure of the pathology is found difficult. We have not used potassium asystole routinely because of the morbidity (increased incidence of heart block) and mortality (inability to restart two hearts† after only brief arrest, of less than 11 minutes) but have preferred to make this decision after the heart has been opened and the pathology inspected.

Results of Open Cardiotomy

These efforts have been directed primarily toward the correction, and even cure, of

† Both patients had aortic valve disease with marked ventricular hypertrophy.

previously inoperable lesions. As a consequence, many of the patients have been seriously ill prior to surgery. Patients with correctable lesions, we believe, deserve the potential benefits of surgical treatment, even though the calculated risk may be high in some seemingly hopeless clinical conditions. For instance, one 8-week-old infant, with only a clinical diagnosis (correct) of ventricular septal defect, was operated upon, using the pump-oxygenator, as an emergency nighttime procedure because the cardiologist was convinced that under medical management she could not survive until morning. Obviously, any group's enthusiasm for accepting these extra-bad-risk cases from among patients uniformly quite ill of their disease will be an important determinant in survival rates. Moreover, much learning was necessary by the inevitably painful method of trial and error. The in vivo problems of unfamiliar or poorly described pathological malformations has demanded on-the-spot improvisations, which, naturally, did not always succeed the first time. These facts are, of course, paramount in evaluating the successes obtained, as well as the failures recorded.

Ventricular Septal Defect.—Ventricular septal defect is a common congenital cardiac malformation, and it probably ranks second only to patent ductus arteriosus in incidence. It has been the most frequent lesion in this series (154 patients). Nevertheless, prior to March, 1954, this defect was incurable, although in the past there has been little question of the desirability of closing ventricular septal defects, particularly those associated with pulmonary hypertension (which includes the vast majority). Of all patients born with a ventricular septal defect, in the times prior to corrective surgery, most died of the consequences of their defect, and many succumb in the first year of life. Those surviving this hazardous initial 12 months dwindle rapidly in number, and only a small percentage reach the teens. Only 2 in this operative series of 154 patients were older than 15 years. Death of the patient, and not factors of case selection, we believe, are responsible for this revealing fact. In our experience many of the older patients were carried in our clinical records for years as cases of "typical" ventricular septal defects, or Eisenmenger's complex. However, upon surgical exploration they were found to have a variant, such as truncus arteriosus, "corrected" transposition of the great vessels, or one of the several forms of single ventricle with or without transposition. The finding of an isolated ventricular septal defect in these older patients certainly occurs, but is unusual. The conclusion is inescapable that a very limited number of those persons born with an isolated ventricular septal defect ever survive beyond the pediatric age group unless cured surgically.

The lethal element of ventricular septal defects is, primarily, the early, rapid, and progressive development of pulmonary hypertension associated with a high pulmonary vascular resistance. From this, it is evident that both the pulmonary pressure and the pulmonary flow must be evaluated to assess the pulmonary vascular resistance. Patients with an increased pulmonary pressure and a high pulmonary blood flow have a low resistance, i. e., are free of significant occlusive arteriolar disease and are good operative risks. On the contrary, those with a high pulmonary-artery pressure but with a modest increase in pulmonary flow usually have a more serious degree of occlusive arteriolar disease, and their immediate hemodynamic readjustment after corrective surgery is considerably more difficult. Marked elevation of the pulmonary vascular resistance has been the most important single factor determining the operative risk.

We believe the most potent stimulus to intimal arteriolar proliferation and medial hypertrophy in a case of ventricular defect is not the increased blood flow through the lungs. Patients with secundum type of atrial defects often have left-to-right shunts of a far greater volume than do those with ventricular defects. Yet the level of pulmonary hypertension is usually lower and appears

TABLE 4.—*Result of Ventricular Septal Defect Repair According to Age and Degree of Pulmonary Hypertension*
154 Cases—71 Male, 83 Female

Age Range, Yr.	No. Patients	Pulm. Artery Systolic Press., No. Pts.	More Than 70% of Systemic Aortic Deaths	Pulm. Artery Systolic Press., No. Pts.	More Than 70% of Systemic Aortic Deaths	Total Mortality
0-1	24	15	9 (60%)	9	3 (33.3%)	50%
1-2	27	12	4 (33%)	15	4 (26.6%)	29%
2-5*	40	26	11 (42%)	14	1 (7%)	30%
5-10	43	21	7 (33%)	22	1 (4.5%)	18.7%
10-20	19	12	4 (33%)	7	0 (0%)	20%
20-38	1			1	1 (100%)	100%
Totals	154	86	35 (40.7%)	68	10 (14.8%)	29.3

* In the 44 patients past the age of 2 years, and with pulmonary hypertension not exceeding 70% of the aortic, there were only 3 deaths (6.8%), and each of these 3 patients had a significant associated lesion.

later. Rather, the initiating and determining factor is the systolic thrust of the left ventricular output delivered undamped to the lung vessels.

Results: One hundred fifty-four patients have been operated upon for closure of a ventricular septal defect. The sex distribution is about equal (71 male and 83 female). As indicated in Table 4, there were 45 deaths, an over-all mortality of 29.3%. The type of by-pass does not appear to have significantly influenced these mortality rates (Fig. 5). However, the adverse consequences of infancy and the advanced degrees of pulmonary hypertension on mortality rates are illustrated in Figure 6. Children under 2 years have been poor operative risks as a group because of the respiratory problems encountered both pre- and postoperatively. The management of secretions, their retention, and the avoidance of pneumonitis are difficult in these tiny infants, often weak and

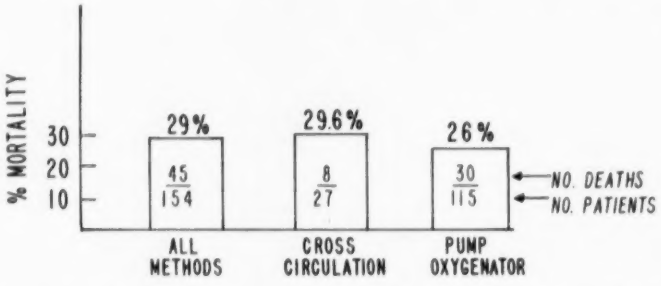

Fig. 5.—Comparison of different methods of extracorporeal oxygenation in patients with ventricular septal defects treated by direct-vision closure.

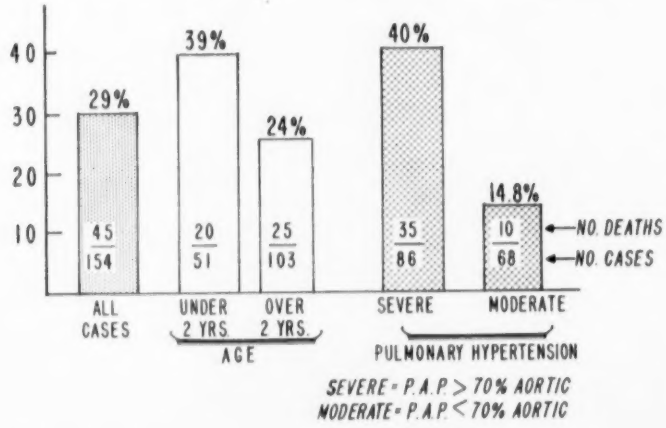

Fig. 6.—Influence of age (infancy) and pulmonary hypertension upon operative risk for patients undergoing ventricular defect closure.

underweight from their cardiopulmonary disease. The muscular development in the chest wall of even healthy infants is poor compared with that found in older children.

The pulmonary pressure is significant only as a reflection of the degree of pulmonary vascular proliferation. Specifically, an individual patient with high pulmonary-artery pressure may have either healthy or severely damaged pulmonary vasculature. Inherent difficulties arise in the accurate measurement of pulmonary blood flow, and the calculations for resistances therefore have less prognostic significance than if they could be precisely measured for the individual patient. On the other hand, a close correlation has been found to exist between the microscopic appearance of the lung vessels and operative risk.[13] If the pulmonary-artery systolic pressure is plotted as a percentage of systemic systolic pressure, the frequency of severe pulmonary vascular changes follows the approach of the pulmonary pressures toward systemic levels. However, it is probable that the incidence of Grade III vascular lung changes does not become universal until these patients demonstrate a reversal of their shunt (at rest). Many patients with ventricular septal defects die of other causes before this state is reached. Thus, the accurate evaluation of operative risk in a particular patient with a high pulmonary pressure from the clinical and catheterization data is often tricky. Lung biopsy, although cumbersome in other respects, has been the most accurate prognostic test in our experience.

Figure 7 portrays further the interplay of these factors of pulmonary hypertension and age upon risk. Noteworthy are the 44 patients beyond the age of 2 years and with considerable, but not advanced, pulmonary hypertension who underwent corrective surgery, with a risk of only 6.8%. In fact, each of these three patients who died had a serious additional defect or lesion responsible for their death, as follows: Case A, aged 2½ years, had a very large left superior vena cava, which was not recognized until after cardiotomy. Considerable blood loss occurred before it was located and clamped. This patient failed to awaken postoperatively, and it is not known for sure whether clamping this large vena cava, with consequent cerebral congestion, or the blood loss was responsible for her neurologic injury. Case B, a 38-year-old woman, died suddenly while out walking six weeks after corrective surgery. Autopsy disclosed that death was due to a coronary thrombosis in remarkably diseased arteries. Her defect closure was completely healed. Case C, a 6-year-old boy, had severe aortic insufficiency with a small ventricular defect. Postoperatively, he was thought to have a ruptured aneurysm of the sinus of Valsalva. At surgery his heart was arrested for only four minutes with potas-

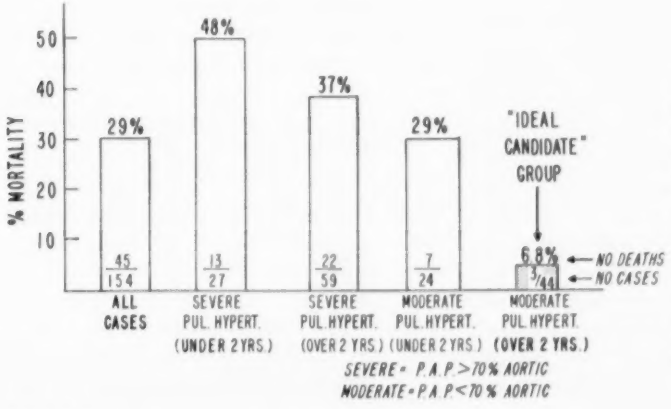

Fig. 7.—Mortality rates according to degree of pulmonary hypertension and age (infancy) in patients undergoing closure of ventricular defects. The pulmonary hypertension is graded on the basis of whether the pulmonary-artery systolic pressure is greater or less than 70% of the aortic systolic pressure.

TABLE 5.—*Other Cardiovascular Anomalies Encountered in 154 Patients with Ventricular Septal Defects*

Lesion	No.
Patent ductus arteriosus	22*
Left superior vena cava	16
Endocardial fibroelastosis	8
Atrial septal defects	6
Multiple ventricular defects	6
Coarctation of the aorta	4*
Mitral stenosis	3
Double aortic arch	1
Dextrocardia	1
Aortic regurgitation	1
Corrected transportation	1

* Three patients had a patent ductus arteriosus and coarctation of the aorta, in addition to a ventricular septal defect.

sium citrate, and it was impossible then to restart it.

The "ideal-candidate group" is presented not with the implication that corrective surgery should be restricted to these fortunate few (28% of the total series) but, rather, to forecast for the future the low risk that may be anticipated for this malformation as the problems associated with pulmonary hypertension are mastered. From Figure 7 it might be deduced that surgery for patients under 2 years of age should best be deferred until they are older. Whether this assumption is valid or not depends on the number dying during this waiting period, and, unfortunately, accurate information on this point is difficult to obtain.

Associated congenital cardiac defects have been frequent among these patients and probably have influenced the mortality to some extent (Table 5).

Indications for Reparative Surgery: Because ventricular septal defects are a common lesion, and because many children die of their defect in the first 12 months of life, a number of the cases seen are of rather desperately ill infants. They suffer from a failure to gain weight, repeated respiratory infections, and recurrent or persistent cardiac decompensations. Virtually all already have severe pulmonary hypertension provoked by mechanisms cited. We consider these persons in urgent need of surgical relief but recognize their increased operative risk. They are often marked for an early death unless successfully operated upon. On the other hand, in the case of an infant with a ventricular septal defect who gains weight at a relatively satisfactory rate and who appears to be responding well to nonoperative management, we would prefer to postpone surgery, if possible, until at least the age of 2 years. The technical aspects of the defect closure during infancy are not excessive. The major hazards hinge on management of their respiratory complications. However, undue procrastination about an operation during early childhood pyramids the risk, for the pulmonary-artery pressures in some patients can double within 12 months. The onset of severe pulmonary hypertension is an ominous sign and indicates that, although correction is still indicated, the most favorable moment for this surgery has passed. The anatomic locations and brief reference to the type of surgical management for the defects in these 154 patients are summarized in Table 6. A more complete description of the anatomy of these defects has been presented previously.[14]

In summary, it is clear that a ventricular septal defect is a most grave congenital cardiac malformation. It is certain to shorten the life expectancy of almost all those persons unfortunate enough to have that problem. Closure of the abnormal opening no longer remains the major problem. For those with mild to moderate degrees of pulmonary hypertension, this can be done with low risk to the patient. Currently, somewhat paradoxically, a greater problem is the development of effective methods for managing their complications associated with advanced pulmonary arteriosclerosis.

TABLE 6.—*Location and Methods of Surgical Repair of Ventricular Septal Defects in 154 Patients*

No.	Location	Ivalon Pledget *	Suture Only	Ivalon Patch †
133	Membranous	58	42	33
11	Posterior	5	3	3
8	Anterior	4	2	2
7	Muscular	3	4	
1	Traumatic		1	
160 ‡		70	52	38

* Held in place by silk sutures.
† Circumferentially stitched in place.
‡ Six patients had multiple ventricular defects.

Postoperative Recatheterization: Recatheterization has been carried out in 36 of the 109 survivors at an interval of 4 to 16 months after operation. In 28 of the 36 patients, no evidence of a left-to-right shunt was found and the defects were deemed closed. Moreover, 25 of these 28 exhibited both a significant drop in the pulmonary-artery pressure (averaging 38 mm. Hg systolic) and a fall in pulmonary blood flow of 6.5 liters per meter^2 per minute (average). The remaining three patients showed no drop in their pulmonary hypertension one year after surgery, although their calculated pulmonary blood flows had decreased. Their pulmonary arteriolar changes may well be progressing, and hence these obliterative arterial processes can be self-sustaining. This evidence can be construed as yet another reason for earlier operations. Unfortunately, all three of these cases occurred early in the series, prior to the time when lung biopsies were more regularly secured. Two of these patients have symptoms. Of the eight patients with demonstrable left-to-right shunts on recatheterization, five now have a normal pulmonary-artery pressure. Clinically they are well, and hence it is likely that the remaining ventricular defect is small. The other three patients each possess a free ventricular communication, with no demonstrable hemodynamic or clinical improvement after surgery.

Thus, 35 of the 38 patients (92%) restudied have benefited substantially from their operative procedure. Most of them were operated upon early in our series. In view of the technical improvements since, the future possibilities are good for a cure rate even closer to 100%.

Atrioventricularis Communis.—This is a complex defect in which the pathologic variations occupy wide spectra. The complete form, encountered in nine patients, consists of an ostium primum atrial septal defect, mitral regurgitation due to a cleft in the aortic leaflet, tricuspid insufficiency because of a similar deformity of the septal leaflet, and a ventricular septal defect from failure of these leaflets to attach firmly to the ventricular septum. This complete form is encountered most frequently in infants because it usually leads to the early development of severe pulmonary hypertension, serious clinical disability, and a shortened life expectancy unless relieved by corrective surgery.

The partial type that we have encountered most frequently (seven patients) has been an ostium primum defect in association with a cleft mitral valve, a normal tricuspid valve, and no ventricular component.

Three patients have had rather a typical form, in which a left ventricular-right atrial shunt arose through a defect between these chambers. It would appear that these instances represent an embryonic variant. The tricuspid septal leaflet was cleft in all, but the mitral was normal in two.

One patient had only an ostium primum without apparent valvar deformity.

The atrioventricularis communis group of defects cannot be corrected by blind or closed methods. Moreover, with the patient under hypothermia, insufficient intracardiac time is available to deal with this complex. With perfusion techniques, however, precise suturing can be accomplished, and cure now becomes possible. Repair consists of closure of the septal defects together with careful correction of the valvar malformations. Our first patient to have these defects (complete form) corrected successfully was operated upon on Aug. 6, 1954, through a right atrial cardiotomy, utilizing controlled cross circulation. The preoperative and postoperative catheterization data upon this infant are contained in Table 7. She has remained asymptomatic in the subsequent years.

Twenty patients, ranging in age from 6 weeks to 29 years, with atrioventricularis defects have been operated upon, with nine deaths. This relatively high risk rate has been compounded from several factors: slowness in development of a clear understanding of the anatomic variables, which permits the surgeon to recognize promptly

TABLE 7.—*Cardiac Catheterization Studies* Before and After Corrective Surgery in Infant with Atrioventricularis Communis Defect*

Site	Oxygen, % Saturation		Pressure, Mm. Hg.		
			Preop.		
	Preop.	Postop.	Quiet	Crying	Postop.
Inferior vena cava	32	79	--	--	--
Superior vena cava	12	75	--	--	--
Right auricle	63	78	3	--	3
Right ventricle	77	70	55/0	90/10	22/4
Pulmonary artery (trunk)	87	78	55/30	90/50	20/4
Femoral artery	95	98.5		135/70	90/50

* Courtesy, Dr. J. D. Keith, Toronto. Preoperative age 6 months (Nov. 9, 1953); corrective surgery Aug. 6, 1954; postoperative studies at age of 19 months (Dec. 3, 1954).

the particular lesion present; the frequent development during the operation of complete heart block, and the advanced disability of some of the patients accepted for surgery. With an increasing experience, some of these ill effects are being overcome. However, three of these deaths were in patients cyanotic at rest. Each had systemic arterial pressure levels in the pulmonary vessel. The treatment of complete heart block by use of isoproterenol U. S. P. (Isuprel) and a pacemaker electrode implanted directly into the myocardium has reduced somewhat the mortality from this vexing complication. We believe, therefore, that even in this small series the operative risk is falling, as evidenced by more successes among recent cases.

Atrial Septal Defects.—The ony acceptable treatment for an atrial (secundum) septal defect is one which produces a cure. It follows, therefore, that the results of all forms of treatment, closed or open, must be appraised after postoperative catheterization. An initial low mortality, while obviously desirable, is not at all meaningful in an ultimate evaluation of the best method for treating this lesion. Most patients with atrial secundum defects, unlike those seriously ill from ventricular septal defects, will survive a thoracotomy and exploratory cardiotomy, even though the opening may not have been completely closed, or even substantially reduced in its diameters. Advocates of the various closed methods for "repairing" atrial septal defects have rarely presented postoperative catheterization data. Such information as is available reveals a substantial incidence[15] of residual shunts. Moreover, another consideration is the difficulty of distinguishing preoperatively in all cases between atrial secundum defects and the various forms of the ostium primum syndrome. Although many authors have described valuable criteria for making this differential, and these signs are of value, suffice it to say that accurate differentiation is not possible in every case despite the considerable skill and experience of these diagnosticians; and the fact remains that in a certain number of patients, when utilizing hypothermia or one of the above-mentioned blind techniques for closure of atrial septal defects, the surgeon will find himself in the operating room with the chest open and at the mercy of the more complicated pathology unexpectedly found to be present.

This is true because it is possible to deal curatively with the atrioventricularis communis defects (ostium primum syndrome) only if one has available a pump-oxygenator and total cardiopulmonary by-pass. With hypothermia or a closed method the patient in this situation is doomed either to death or, at best, if he survives, to a thoracotomy which not only serves no purpose but actually impairs his chances for successful curative surgery at a later time, using cardiac by-pass, because of the hazard induced through bleeding from vascular adhesions when he is reoperated upon and heparinized. These considerations are sufficiently vital to the patient's well-being, and we advocate the use of total cardiopulmonary by-pass with the pump-oxygenator for repair of all atrial septal defects. The dividends of this policy

have been numerous. First, in a small series of 12 patients, ranging in age from 2 to 51 years, with atrial secundum defects there have been no deaths. The ability to close even these simple defects in an unhurried and precise manner with interrupted stitches indicates that the percentage of anatomic cures may be expected to approach 100%. Finally, we have encountered unexpectedly complicated secundum defects that could be repaired completely with ease. In one such patient with unsuspected drainage of the right pulmonary veins into the superior vena cava and a foramen ovale defect, the repair, although straight forward, required a by-pass interval of 55 minutes, to avoid encroachment by the repair upon these venous channels. Moreover, we have encountered an ostium primum lesion in several instances in which even after retrospective study there was no reason to suspect more than a secundum lesion.

It is quite predictable that other cardiologists and surgeons will, as they acquire confidence in their pump-oxygenator, abandon completely the less satisfactory methods that have been advocated in the past for closure of atrial septal defects.

Tetralogy of Fallot

The Blalock-Taussig anastomotic operation and the Potts modification have, in the decade or more since their introduction, brought effective palliation to the lives of many previously crippled from the physiologic abnormalities associated with Fallot's tetralogy.

Inevitably, however, because this procedure is by definition palliative, the mortality and morbidity continue to rise at an abnormal rate with the lapse of time. The magnitude of this increased attrition was charted in a follow-up study recently completed for all of the patients having the systemic-pulmonary artery shunt performed at the University of Minnesota Hospitals (Table 8). Whereas the initial (operative) mortality rate of 9% was about standard, the late deaths (21%) have more than doubled

TABLE 8.—*Tetralogy of Fallot* * *Systemic-Pulmonary Shunt* University of Minnesota Hospitals 1946-1954

Patient Status January, 1957			110
Total patients			
Died of operation	10 (9%)	30%	
Died later	23 (21%)		46%
Unimproved	18	16%	
Improved	62		54%

* Tricuspid atresia, single ventricle; transposition cases excluded.

this primary risk. Furthermore, at the time of their check-up, from 2½ to 10 years after operation, there were 30% who were dead, and another 16% were in need of further surgery.

These late failures recorded with the palliative procedure were most frequently not related to the closure of a previously functioning shunt but, rather, were associated with the patient "outgrowing" his shunt. This same fact explains why the results of the shunt operation have been generally poorer when performed either in infants or in adults.

The fact that 54% of this series still remain improved is certainly good evidence of the palliation that was provided by the shunt procedure. Its merit is even more apparent when one recalls the outlook for these persons prior to its offering. Rather, these studies have served to define for us the area in which improvement is both necessary and possible.

The basic defects in Fallot's tetralogy are twofold—ventricular septal defect and pulmonary stenosis. Clearly, the goal in these patients is the complete correction of all existing defects by open cardiotomy. After leaving this circulation restored to normal anatomically and physiologically, one can anticipate that life expectancy may be lengthened to its full span. Moreover, it is likely that the total risk for this patient undergoing curative surgery is limited to the initial operative hazard.

Since August, 1954, at the University of Minnesota Heart Hospitals, we have not found it necessary to perform any shunt procedures in tetralogy of Fallot cases. All

patients deemed in need of surgical assistance have had the curative procedure (closure of the ventricular septal defect together with pulmonary infundibular resection and/or pulmonary valvulotomy).

The operative indication for the completely corrective procedure has been similar to those recognized as valid for the shunt procedure and consists of definite evidence of deterioration. This state may be represented either by recurrent bouts of heart failure (usually in infants) or by decompensation of the pulmonary collateral channels (syncope episodes, extremes of dyspnea upon slight effort, or a sustained rate of increase in the hemoglobin level, erythrocyte count, or hematocrit value).

These patients include a number of infants, and the youngest, a success, was 16 weeks old.

With the curative procedure the operative risk has steadily decreased as experience has been gained with the technical problems involved. It is likely that eventually the operative risk among tetralogy cases will be considerably less than that for simple ventricular septal defects, because the pulmonary circulation in the tetralogy group has not been damaged by pulmonary hypertension. In that regard, a tetralogy patient with a left-to-right shunt already represents a low surgical risk. Furthermore, the corrective procedure is technically feasible at all ages, from infancy to adult life (our oldest patient, also successful, was 48). We have been gratified with the results of postoperative studies in which a drop was found in the right ventricular pressure without a gradient, or with a very small gradient across the infundibular or valvular area into the pulmonary artery. In the 40 tetralogy patients of this series the mean postoperative femoral-artery oxygen saturation was 93.7% (87% to 100%), as compared with the mean preoperative value of 51% (19% to 86%).

For the patient with tetralogy of Fallot defects in areas where facilities for open-heart surgery are not yet available, we currently recommend withholding, save for the most desperately deserving, any shunt procedure, with the hope that a corrective operation can be carried out eventually. This philosophy does not deny the risk of complications for certain of these patients. Rather, it stems from experiences which indicate that the creation of a shunt procedure has enhanced greatly the patient's risk when intracardiac surgery is subsequently attempted (Table 3). The bleeding problems from these extensive collaterals and arteriolar adhesions which develop in cyanotic patients who have had thoracotomies are indeed formidable during and after heparinization.

Acyanotic Tetralogy.—This group includes patients with pulmonary stenosis, either valvar or infundibular, plus a left-to-right shunt at the ventricular level. Their pulmonary stenosis is mild compared with that in the cyanotic variety and is usually, but not invariably, infundibular. They are, in effect, "protected" by the pulmonary stenosis from the consequences of their ventricular septal defect. We have operated upon six of these patients, with one death. The latter was an infant (11 months of age) with pulmonary stenosis. Preoperative cardiac catheterization and angiography revealed no evidence for a septal defect at either the atrial or the ventricular level. At operation, a pulmonary valve stenosis was found and corrected; the ventricular defect was overlooked, even though the ventricle had been opened. The baby died 12 hours later, and autopsy revealed a large (12 mm.) ventricular defect concealed between hypertrophied trabeculae carnae.

Pulmonary Stenosis with Intact Ventricular Septum.—Pulmonary stenosis with an intact ventricular septum has been encountered in various forms among the 15 patients operated upon by open-heart perfusion methods. Except for the atresia group, this has been a satisfactory lesion for surgery by this method. When the blind transventricular approach has been used, an acceptable immediate operative mortality was possible. Unfortunately, follow-up studies

indicated that 30% to 40% of the patients remained uncured, as judged by hemodynamic measurements. The reasons for this are twofold: (1) incomplete or inaccurate division of the pulmonary valve; (2) failure to recognize or to be able to correct associated defects (infundibular stenosis, or atrial or ventricular septal defects).

Hypothermia permits an accurate opening of the narrowed pulmonary valve. Insufficient time is available, however, for correction of some of the associated defects that are frequently encountered unexpectedly. If a ventriculostomy is necessary, the heart is intolerant of this maneuver. The best approach to achieving the ideal of complete cure in patients with pulmonary stenosis is through use of a by-pass method with a pump-oxygenator.

The atresia group with intact ventricular septum, however, has remained an unsolved problem for us. All of the five infants, under 6 months of age and cyanotic, succumbed. Surgical intervention was often used as an emergency, for their existence was so precarious. The problem appears to be a physiologic one. The thick-walled, small cavity of the right ventricle is relatively nonfunctioning. In each case, despite relief to the outflow-tract obstruction under direct vision, the ventricular myocardium seemed unable to adjust from an isometric to an isotonic type of contraction. Death ensued within a few hours. Cases of pulmonary atresia associated with a ventricular septal defect are less prone to present this problem.

Total Anomalous Pulmonary Venous Connection.—This rather serious and relatively uncommon malformation can now be cured through open-heart methods. Those patients with veins entering directly into the right atrium present no particular problem, since they do not differ dynamically from persons with a large atrial (secundum) defect. However, in those with total anomalous connections to the superior cava, coronary sinus, or elsewhere the left side of the heart fails to develop. The adjustments required after complete correction can provoke considerable pulmonary edema. Doubt has been voiced that a one-stage reconstruction can be carried out in these patients. More cases are required for certainty. However, currently we believe that if great care is exercised in the administration of blood and fluids during and after the operation, successful results are possible. After our recognition of this need, the last case did well. One of our failures was due to pulmonary edema, which we feel now might have been preventable, and the other was from an avoidable technical error (Table 3).

Complete Transposition of the Great Vessels.—This common congenital malformation, invariably fatal in infancy, continues to remain a major challange to cardiac surgeons. After a modest experience with palliative operations,[16,17] we abandoned that concept in favor of total correction of all the primary and associated defects. This required a by-pass and the pump-oxygenator. So far, success has eluded us, as it has others; yet someone with superior ideas is certain soon to identify the proper techniques for overcoming these difficulties.

Ruptured Sinus of Valsalva.—Rupture of an aneurysm of the sinus of Valsalva into one of the cardiac chambers results in a left-to-right shunt. An added consequence is a reduction in the systemic diastolic pressure. The characteristic murmur and findings at catheterization make the correct diagnosis relatively likely. Until recently persons suffering from this lesion either died prematurely or were incapacitated by their progressive cardiac decompensation. Since May 2, 1956, three patients (aged 6 to 37 years) with this malformation have been operated upon, utilizing the pump-oxygenator. Successful corrective surgery has been possible in all cases. Retrograde perfusion of the coronary sinus was used in one, and elective potassium asystole in the other two, to permit cross clamping the ascending aorta.

Aortic Stenosis.—Patients with congenital aortic stenosis requiring surgical relief are best treated by an open method utilizing total cardiopulmonary by-pass and the pump-

oxygenator. When the congenital aortic lesions were operated upon with closed methods, the results were often poor. Aortic insufficiency was produced in those with valvular lesions, and the surgeon found that adequate relief of any infundibular obstruction of the subvalvar type was difficult to achieve. In our own experience, these subvalvar intrusions have occurred in two out of the three cases.

The direct-vision exposure of the valve with acquired aortic stenosis offers promise of improving the results of aortic valvotomy. A more effective placement of the commissurotomy and some freeing of adhesions to the sinuses can be accomplished to mobilize the cusps. However, some valves are so severely destroyed by their calcific disease that reconstruction is not possible, and really effective valvular function may not be realized until a satisfactory prosthesis is developed which can be inserted proximal to the coronary ostia.

Mitral Regurgitation.—Under direct vision and with the pump-oxygenator, we have operated upon two patients, each completely incapacitated preoperatively by pure mitral regurgitation. The lesion then could be carefully studied and repaired. Good surgical exposure of the mitral valve was secured through a right lateral thoracotomy and a left atriotomy. Mattress sutures of silk were used to plicate the annulus in order to permit opposition of the leaflets to the point of sufficiency. By tying these stitches down on partially compressed pillows of polyvinyl formalinized (Ivalon) sponge, the spinnaker-like action of the aortic leaflet achieved greater competency as well. Both patients recovered and have been impressively improved. The first patient, operated upon Aug. 29, 1956, sustained a drop in left atrial pressure from 43/26 to 18/10 mm. Hg immediately after corrective surgery.

Mitral Stenosis.—The use of an open approach to mitral stenosis will remain controversial until a wholly satisfactory replacement is available for these damaged leaflets. However, the surgeons with great experience in this operation have emphasized that mortality in patients with Stage IV disease is many times higher than that in Stage II and Stage III cases. The need for improvement here is thus self-evident. Apropos of this point, we have selected for mitral valvuloplasty under direct vision those patients who have reached Stage IV in their clinical course. This admittedly small experience suggests that the surgeon can accomplish a more satisfactory operation by the open method. It has also provided a satisfactory solution to the vexing problem of preoperative estimations of just how much insufficiency and/or stenosis a particular patient has. In these cases, whether stenosis or regurgitation exists, both can be managed while the auricle is open.

Summary and Conclusion

A variety of progressively disabling cardiac lesions, both congenital and acquired in origin, and in many instances previously inoperable, have been treated surgically among the 305 patients submitted to open cardiotomy, at normal body temperatures, and utilizing an extracorporeal circulation.

Although both the techniques and the knowledge of the physiology of total body perfusion in man had to be developed and despite the fact that a number of new surgical procedures had to be devised and tested, the risks have not been discouraging when examined in terms oftentimes of significant improvement in otherwise hopeless conditions.

Of these 305 patients, ranging in age from 6 weeks to 66 years, 204 (67%) survived. A majority of them are completely cured, thanks to the accuracy that visual methods give to surgical technique. Moreover, a study of 101 failures, early and late, leads to a realization that the increased knowledge thereby derived will assist in preventing similar types of failures in the future.

Open-heart operations are a surgical endeavor likely to continue to grow steadily in stature. The benefits are very real to those patients requiring this help.

Beyond these obvious surgical applications, the use of a pump-oxygenator in the treatment of otherwise irreversible severe shock from trauma, blood loss, burns, etc.; for toxemias of various types, extensive pulmonary disease, or edema, and to rest the heart in cases of severe infarctions or decompensated states remains virtually an unexplored promise. In some of these situations an artificial heart-lung-kidney quite likely could be life-saving.

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The Postoperative Radical-Mastectomy Syndrome

Its Treatment with Stellate Ganglion Block

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The postoperative radical-mastectomy syndrome is caused by the extensive surgery necessary in the efforts to cure a malignancy of the breast. The most characteristic sign of this syndrome is edema of the arm, but, in addition, one or more of the following signs and symptoms are usually present: accumulation of fluid under the skin flaps and in the axilla, delayed wound healing, pain in the arm and/or the chest, lymphangitis, induration of the arm and/or the chest wall, limitation of motion of the shoulder girdle and the arm, and hypertrophic cicatrix formation (Fig. 1).

The following factors, either individually or in combination, have been cited as the cause of the edema of the arm and could be responsible for the other signs and symptoms of the syndrome: (1) venous obstruction; (2) lymphatic-vessel destruction; (3) infection; (4) mechanical obstruction of the vessels by scar formation, metastatic tumors, and/or fat fold; (5) irradiation therapy; (6) skin grafting, and (7) removal of axillary vein during the operation.[1-13] While one of these factors or a combination of them may or may not be the cause of this syndrome, any one of them can produce a chronic vasospasm.[14] Chronic vasospasm, regardless of its cause, tends to perpetuate itself and often becomes worse; i. e., it sets up the so-called "vicious cycle" (Fig. 2). Thus chronic vasospasm could in many cases be the sole cause of the postoperative radical-mastectomy syndrome.

The efficacy of stellate ganglion block in correcting chronic vascular spasm and thereby reducing the swelling and the edema of the arm following radical mastectomy has been shown. Hanelin, Williams, Wolffson, and Wernick[15] injected the stellate ganglion in 25 cases of edema of the arm and reported persistent subjective improvement in 80% of the patients. Feldman[16] advocated daily paravertebral blocks of the lower cervical and upper thoracic sympathetic ganglia to prevent postoperative edema of the arm following a radical mastectomy. Arnulf[17] supported stellate block therapy and stated:

> I have personally done many infiltrations for edema of the upper limb after Halsted operations. In general the edema diminishes without completely disappearing; the pains, when they exist, are calmed quite regularly. Despite the inconsistency of these results, I believe it legitimate to use stellate infiltrations in all cases; no patient has been discommoded by them, and even those whose edema is not influenced feel a limb more supple and more usable after infiltration.

Our paper reviews the use of stellate ganglion block in 100 patients whose postoperative course following radical mastectomy was complicated by the postoperative radical-mastectomy syndrome.

Method of Study

Patients who showed signs and symptoms of the postoperative radical-mastectomy syndrome following removal of the breast received a series of stellate ganglion blocks, i. e., 2 to 12 blocks. With the exception of the first few blocks, which on occasion were given to the patients while they were still in the hospital recovering from the radical mastectomy, most patients received the block therapy as outpatients.

The anterior (paratracheal) approach, which is fully described in other publications, was selected as the technique of choice for executing the block

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Fig. 1.—Patient with typical postoperative radical-mastectomy syndrome. Note tight, fixed skin; swollen arm; hypertrophic cicatrization, and drainage site. Motion of arm was limited. Illustration reproduced from "Stellate Ganglion Block," by D. C. Moore,[19] published by Charles C Thomas, Publisher, Springfield, Ill., 1954.

because it is simple for the physician to master and is not a painful procedure for the patient.[18,19] It consists essentially of having the patient lie in the dorsal recumbent position without a pillow and with her head tilted backward so that the neck is in maximum extension. The transverse process of the seventh cervical vertebra is determined, and the skin overlying it is marked with an *X* along the medial border of the sternocleidomastoid muscle. The area is aseptically prepared and draped. A skin wheal is made. A 1½ or 2 in. (3.8 or 5 cm.), short-beveled security bead needle is inserted through the skin wheal and pushed posteriorly until it impinges on the transverse process of the seventh cervical vertebra. The point of the needle is then withdrawn ⅛ in. (0.3 cm.) and, after precautionary tests are executed to assure that the bevel of the needle does not rest intravascularly or subarachnoidally, 10-15 cc. of the local anesthetic solution is injected and the needle withdrawn. If this method of blocking the stellate ganglion is correctly executed, paralysis of ganglia of the cervicothoracic portion of the sympathetic nervous system on the side blocked results.

Within 2 to 30 minutes following the block a Horner's syndrome and evidence of sympathetic block of the upper extremity and chest wall will appear. It should be remembered by the physician executing the block that if the sympathetic chain is blocked at any place in the neck, a Horner's syndrome (ptosis, enophthalmos, and miosis) will appear. He may have blocked the cervical portion of the cervicothoracic chain but not the thoracic portion. This makes little difference if the block was performed for a sympathetic block of the head and its contents; but if it was executed for disease in the upper extremity or chest, no benefit will be derived. This point is stressed because all too often the physician will be satisfied that he has executed a stellate block if a Horner's syndrome appears. Whenever a stellate ganglion block is used as therapy for the postoperative radical-mastectomy syndrome, a careful check of the upper arm should always be made for the signs of a sympathetic block, i. e., increase in skin temperature, dryness, and vasodilatation of the peripheral veins. If these are not present, the block should be repeated, careful attention being given to each step of the technique, particularly the location of the landmarks. However, at least 15 to 30 minutes should elapse before the block is repeated. It is not uncommon for a Horner's syndrome to appear in 2 to 5 minutes after the block has been completed, but it may take 15 to 30 minutes for the arm to show signs of an adequate sympathetic block.

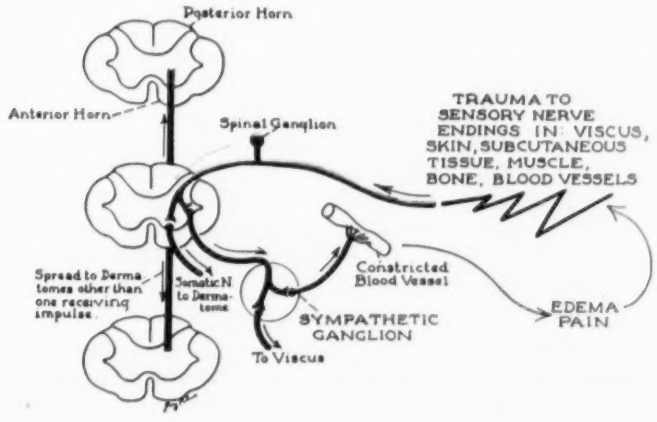
Posterior Horn
Anterior Horn
Spinal Ganglion
TRAUMA TO SENSORY NERVE ENDINGS IN: VISCUS, SKIN, SUBCUTANEOUS TISSUE, MUSCLE, BONE, BLOOD VESSELS
Spread to Dermatomes other than one receiving impulse.
Somatic N to Dermatome
Constricted Blood Vessel
EDEMA PAIN
SYMPATHETIC GANGLION
To Viscus

Fig. 2.—Schematic drawing of the "vicious cycle." Illustration reproduced from "Stellate Ganglion Block," by D. C. Moore,[19] published by Charles C Thomas, Publisher, Springfield, Ill., 1954.

Results

One hundred patients, whose ages ranged from 35 to 78 years and whose signs and symptoms had been present from one week to five years following surgery, received 2 to 12 stellate ganglion blocks.

Sixty-three of the patients showed marked improvement, while twenty-one of the patients showed moderate improvement. Sixteen of the patients showed no improvement. In the last group of 16, however, only 9 submitted to the prescribed series of blocks, i. e., approximately 10 blocks. The remaining group would submit to only four blocks or fewer.

Comment

It is our belief that chronic vasospasm plays a definite, if not the essential, role as a causative factor in the postoperative radical-mastectomy syndrome. If this vascular spasm is not corrected early, fibrosis or scarring may hold the blood vessels in a chronic state of diminished caliber. The action of stellate ganglion block in these cases is threefold. First, it tends to eliminate the vasospasm caused by the surgery, infection, or accumulation of fluid, and this, in turn, probably reduces the pain. Second, it increases what is left of the blood supply to the skin flap and the chest wall after the blood vessels of the breast have been removed. The increased blood supply softens the old scar or keeps the new scar supple, aids in preventing a slough of the skin flap, helps prevent infection, and aids in correcting it if present. Finally, any form of active therapy is a good morale builder, particularly when improvement from it is noticeable to the patient. These patients have undergone a surgical procedure which is mentally traumatizing. They have lost a breast and have been told, or realize, that cancer was present. If, in addition to this, the wound is not healing satisfactorily and the other signs and symptoms of the syndrome are present, these patients may become more discouraged.

A single stellate ganglion block in the postoperative mastectomy syndrome is seldom indicated as a diagnostic procedure to see whether this type of therapy will be efficacious. The results of a single block, or even three to four blocks, are seldom dramatic. From our series of cases, we have come to believe that unless the patient is willing to submit to from 7 to 10 blocks, stellate ganglion block therapy should not be instituted. Furthermore, we never promise the patient anything. We merely tell her about our previous results or have her discuss the therapy with a patient who has had a series of the blocks.

Only 7 of 100 patients who submitted to stellate ganglion block therapy refused to continue the treatment after four or less blocks. Cost of the block procedure was not a factor in these cases, because if any charge was made it was for the block tray only ($2.50). The physician performing the block made no charge for his service—it was considered a part of the postoperative care of these patients. This was explained to the patient prior to the initial block. The refusals were due to the minor complications which may accompany the block procedure, i. e., (1) pain while the needle is being placed, (2) hoarseness from overflow of the local anesthetic solution onto the vagus or recurrent laryngeal nerve, (3) Horner's syndrome, and (4) paralysis of the arm from overflow of the local solution onto the brachial plexus. These minor complications have caused the average patient no concern. All patients were warned that if pain occurred it would last for only a few seconds and that if the other minor complications ensued they would automatically be corrected in a matter of a few hours, as the local anesthetic solution was dissipated. If hoarseness occurs, the patient should be instructed not to eat or drink while it is present. Hoarseness indicates that the vocal cords, the "watchdog" of the trachea, are partially ineffective; and when they are in this condition, food or fluid may enter the trachea easily.

To date we have not seen a single serious complication, i. e., high-spinal, severe systemic toxic reactions, etc., following a stellate ganglion block when the anterior (paratracheal) approach was used.[18] Nonetheless, equipment and drugs for resuscitation, i. e., means of giving oxygen by bag and mask, intravenous fluids, airways, vasoconstrictor drugs, suction, and short-acting barbiturates, have been immediately available.

The following methods of administering stellate ganglion blocks in the patient with a postoperative radical-mastectomy syndrome have been evolved through trial and error.

Immediate Treatment of Post-Radical-Mastectomy Syndrome (i. e., within the first week postoperatively).—Postoperatively the patient's arm should be carefully watched, as the edema of the arm is usually the first sign of trouble. If the edema does not subside within one or two days, the dressing is loosened. If this does not produce the desired results, then stellate ganglion blocks should be begun, at one- or two-day intervals for four to six blocks. After this, the blocks should be given once or twice a week until 10 blocks have been given. The results in the occasional case may be dramatic after the first block. Often, however, the effectiveness of the therapy cannot be evaluated until 5 to 10 blocks have been given. Regardless of the initial result, a series should be performed.

If x-ray therapy is used to supplement surgery in a case in which a series of stellate ganglion blocks has been completed, then the patient should receive one block a week during the period of the x-ray therapy and for two weeks following its completion. The x-ray therapy may cause a brawny induration with accumulation of fluid, and if this is allowed to organize, little, if anything, can be done to reverse the enlargement of the arm.

When stellate ganglion block therapy is instituted early, as described here, the edema of the arm may be markedly reduced. The impression should not be given that the swelling will decrease until the arm has the same measurements as the arm on the intact side. In some cases the arm may be reduced to its normal size, but in others some residual swelling remains, usually not adding more than one inch to the circumference of the upper arm. Most women will not complain of this difference, as it is hardly noticeable. They can wear short-sleeved dresses or long-fitted-sleeved dresses without having the sleeve altered. It is interesting to note that the edema which remains after block is almost entirely confined to the upper arm. The forearm and the hand usually return to normal. The skin edges heal more rapidly, and there is little or no slough of the skin flap. If the wound is draining and/or infected, it is not uncommon for the drainage to decrease or stop and the infection to clear during stellate ganglion therapy.

The patient must be encouraged to move her arm. Regardless of whether stellate ganglion block therapy is instituted early or late, no restrictions should be placed on the use of the extremity. The patient should merely be instructed to keep the arm slightly elevated when she is lying down or sitting in a chair. This tends to promote drainage of lymph and blood, whereas a dependent position of the arm would hinder drainage. If the patient limits the use of the arm, then physical therapy and exercises (for example, the one described under "Delayed Treatment") must be instituted.

Delayed Treatment.—If stellate ganglion therapy is delayed for one to six months while other therapy is being tried instead of being used in conjunction with the other therapy from the outset, the results are not as dramatic; but even when it is delayed, the value of this therapy justifies its use. In these cases the block should be given once or twice a week for 10 treatments. If, however, at the end of 10 injections the following described improvement has not occurred, further stellate block therapy is seldom indicated.

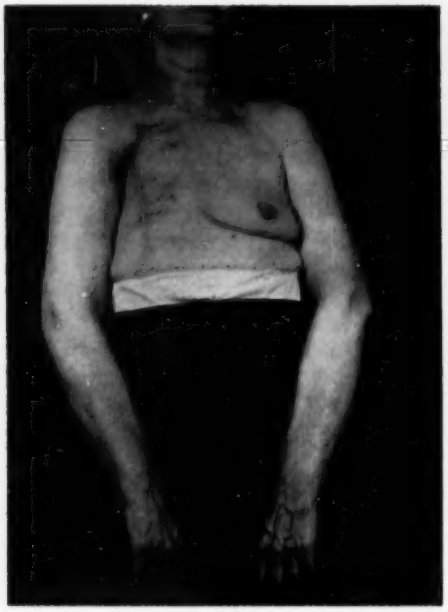

Fig. 3.—This patient, when first seen five months following a radical mastectomy for cancer, had pain and brawny (hard) edema of the arm. She would not move her arm away from her side. Illustration reproduced from "Stellate Ganglion Block," by D. C. Moore,[19] published by Charles C Thomas, Publisher, Springfield, Ill., 1954.

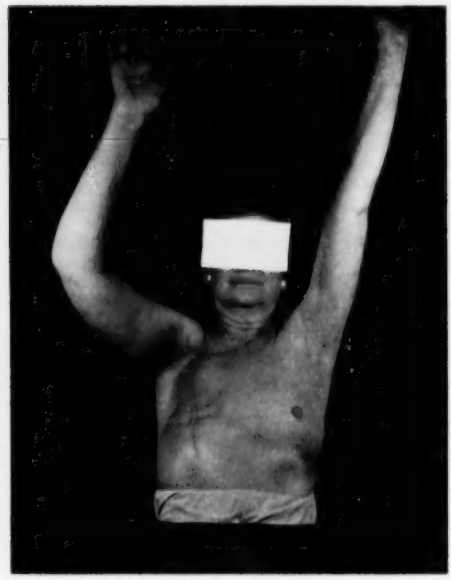

Fig. 4.—Same patient as in Figure 3 after six stellate ganglion blocks, combined with the special exercise described herein. Illustration reproduced from "Stellate Ganglion Block," by D. C. Moore,[19] published by Charles C Thomas, Publisher, Springfield, Ill., 1954.

In most instances the edema will be improved. Even if the size of the arm does not decrease, the brawny induration disappears, and the arm becomes supple. The dragging sensation from the swollen arm likewise disappears. If the wound has been draining, granulating, or infected, one is amazed at the rapid improvement. The block in most cases will speed granulation, slow drainage, clear the infection, promote epithelization, and in general prepare the area for skin grafting if it is necessary. The dull, aching pain, which becomes severer with daily chores, such as housekeeping, is greatly improved, and in many cases will disappear.

The extent of motion of the arm is often impaired in these cases (Fig. 3). With the block therapy and physical therapy, full motion may be restored (Figs. 4 and 5). It is mandatory in this type of case to have the patient exercise her arm. This may be accomplished by having her do her own housework, as well as special exercises, or it may require supervised physical therapy. One of the easiest and best exercises is to have the patient stand next to an open door or wall and, by using her fingers, slowly walk the arm up the door until eventually the hand reaches the top of the door or a clothes hook. The patient then grasps the top of the door or a clothes hook and bends her knees slightly, stretching the arm. In many instances, within two to five weeks after the institution of exercises and blocks, the patient will say, "Doctor, I am amazed at how I no longer favor my operated arm. I reach for objects unconsciously with it, where before I protected it and seldom used it."

In patients who have had edema of the arm with pain for longer than a year and whose wound has healed, little, if anything, can be done about the swollen arm. Usually the pain is relieved and motion improved by blocks. These improvements may be only temporary and may last one or two weeks

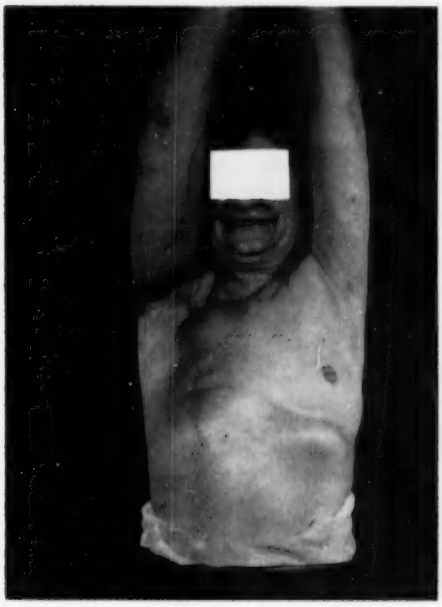

Fig. 5.—Same patient as in Figures 3 and 4, one week after completing a series of 10 blocks combined with the exercise. The patient's arm had not decreased markedly in size, but the pain had disappeared. She had full motion of the arm, and the arm was soft. Illustration reproduced from "Stellate Ganglion Block," by D. C. Moore,[19] published by Charles C Thomas, Publisher, Springfield, Ill., 1954.

at a time. However, in the exceptional case a most astounding result, with decrease in the swelling, softening of the cicatrix, and disappearance of pain, may be obtained. This often justifies the use of the block in these patients, provided they thoroughly understand that improvement may not occur.

In patients who have had stellate blocks and have derived an excellent result, occasionally the swelling and edema may return after the performance of an unusual task, such as having the hand in ice water for two or three hours while freezing fruit. In these cases two to five stellate ganglion blocks will usually correct the edema and pain.

Although many patients with this syndrome improved with good medical and surgical treatment alone, e. g., meticulous care of the wound, drainage of accumulated serum, and maintenance of the general physical and nutritional status by vitamins, blood transfusions, physical therapy, etc., who can predict in which patients the edema of the arm, which is the cardinal sign of the syndrome, will be permanent and in which patients it will be transitory? We agree with Arnulf [17] that no patient has been discommoded by stellate ganglion block therapy and that even if the edema of the arm is not markedly reduced, the patient usually feels that the arm is more supple and usable after the infiltration. Since all therapy which may be of benefit should be offered the patient with the postoperative radical-mastectomy syndrome, we believe that a series of 7 to 10 stellate ganglion blocks should be included.

Summary

Stellate ganglion block was used in 100 women who, subsequent to a radical mastectomy, had edema of the arm with or without one or more of the following: accumulation of fluid under the skin flaps and in the axilla, delayed wound healing, pain in the arm and/or chest, lymphangitis, induration of the arm and/or chest wall, limitation of motion of the shoulder girdle and the arm, and hypertrophic cicatrix formation. After a series of seven or more such blocks, 63% of the patients showed marked improvement, 21% of the patients showed moderate improvement, and the remaining 16% showed no improvement.

On the basis of this series, we believe that stellate ganglion block therapy should be instituted, together with medical and surgical treatment, for patients who exhibit the above-noted signs and symptoms of the postoperative radical-mastectomy syndrome.

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Newer Methods in the Diagnosis of Hyperparathyroidism

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The diagnosis of hyperparathyroidism is dependent on the laboratory detection of chemical changes which occur in the blood and urine when this condition is present. The definitive treatment of this condition requires surgery. Unless it is treated, debilitating or fatal complications, especially due to renal damage, result. Therefore dependable diagnostic laboratory tests are important. In recent years several tests which promise to provide more definite diagnostic criteria for this condition and to permit the diagnosis of early and mild forms of the disease have evolved.

Problems in the Diagnosis

Traditionally, the diagnosis of hyperparathyroidism has been based on the following findings:

1. Either osteitis fibrosa cystica or renal calcification in the form of stones or nephrocalcinosis. Osteitis fibrosa cystica is uncommon in this country at the present time because our diet is high in calcium and phosphorus. The number of diagnoses of hyperparathyroidism established in various clinics in this country has been commensurate, for the most part, with the alertness with which patients with renal lithiasis have been evaluated.

2. Levels of serum calcium over 11 mg. per 100 cc. and a serum phosphorus under 3.5 mg. per 100 cc.

However, the findings are often equivocal, so that a positive diagnosis of primary hyperparathyroidism is at times very difficult. Among the problems that arise are the following:

1. There is difficulty in obtaining accurate and consistent determinations of phosphorus and, especially, of calcium. There are technical difficulties in these determinations. Also, levels of serum calcium and phosphorus vary from time to time in a given subject, especially with regard to dietary variations.

2. Normal levels of serum calcium and phosphorus have been demonstrated in patients with proved hyperparathyroidism without renal insufficiency. In 1932 Albright, Bauer, Claflin, and Cockrill [1] showed that in patients with hyperparathyroidism a very high intake of phosphate would abolish the chemical manifestations of hyperparathyroidism.

3. The clinical manifestations of hyperparathyroidism are sometimes unusual and bizarre, as pointed out by Lee, McElhinney, and Gall.[2]

4. Other conditions will produce variations in serum calcium and phosphorus levels which simulate those which occur in hyperparathyroidism. These include hypervitaminosis D, sarcoidosis, osteomalacia, and certain other bone diseases.

5. The differentiation of primary hyperparathyroidism with secondary renal damage from primary renal disease with secondary hyperparathyroidism, such as the Burnett syndrome,[3] may be very difficult.

It is because of these difficulties that we have been interested in several newer tests for hyperparathyroidism.[4] These tests are based on the physiologic action of the parathyroid hormone. Although the functions of the parathyroid hormone are incompletely understood, it is established that it does increase renal excretion of phosphorus.

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Phosphorus-Reabsorption Test

In the phosphorus-reabsorption test, also termed the tubular reabsorption-of-phosphate test, parathyroid function is evaluated by measuring this primary action of the parathyroid hormone.[5,6] The percentage of phosphorus present in the glomerular filtrate which is reabsorbed by the renal tubules is determined. The normal value for tubular reabsorption is in the vicinity of 90%, whereas this value is approximately 60% in patients with hyperparathyroidism. The precise range for normal subjects has not been established; Chambers and associates[7] report variations of 78% to 90% for normals and 41% to 82% for patients with hyperparathyroidism. After surgical removal of parathyroid adenomata, the reabsorption of phosphate increases to the normal level.

The technique that has been used for the test has been to place the patient on a diet containing moderate and constant amounts of calcium and phosphorus for three to five days. Fasting urine collections are then obtained for periods of several hours, and blood is withdrawn midway in the collection period. Both the blood and the urine specimens are analyzed for phosphorus and creatinine. The glomerular filtration rate is obtained from the determination of the endogenous creatinine clearance. Then, utilizing the values for serum and urinary phosphorus, the tubular reabsorption of phosphorus is calculated:

$$C_{CR} \times \text{serum P} = GFP$$
$$GFP - \text{urine P} = TRP$$
$$TRP/GFP \times 100 = \%TRP$$

C_{CR}=creatinine clearance, ml/min.
GFP=glomerular filtration phosphorus, mg/min.
TRP=tubular reabsorption phosphorus, mg/min.

Chambers and associates[7] use the following formula for their calculation of tubular reabsorption of phosphorus:

$$\%\ TRP = 100\left(1 - \frac{UP \times SC}{UC \times SP}\right)$$

UP-urine concentrations of inorganic phosphorus, mg/ml.
SC-serum concentration of creatinine, mg/ml.
UC-urine concentration of creatinine, mg/ml.
UP-urine concentration of inorganic phosphorus, mg/ml.

Experience with this test thus far has been encouraging. It appears to be reliable. Goldman and associates[8] report a positive test in 20 of 21 patients with hyperparathyroidism over two years. In our experience it appears to be an easy test to carry out and gives a clear-cut differentiation of the normal from the hyperparathyroid. It is of definite help in making the decision for or against surgery.

Insufficient experience has been gained thus far by which to evaluate the test in patients with the milk-alkali syndrome, in secondary hyperparathyroidism due to other causes, in various other conditions of disturbances in calcium and phosphorus metabolism, and in renal damage due to various causes. We found the test to be normal in a patient with secondary hyperparathyroidism associated with osteomalacia of unknown etiology. Chambers and coworkers[7] found the test to be valuable in patients with osteoporosis and also in patients with renal lithiasis but without kidney damage.

Intravenous Calcium-Tolerance Test

It has been known that serum phosphorus levels rise and the urinary excretion of phosphorus decreases in the presence of hypercalcemia. Howard, Hopkins, and Connor[9] incorporated these findings in a test for hyperparathyroidism termed the calcium-load test, or the intravenous calcium-tolerance test.

In this test hypercalcemia is produced by the intravenous administration of a calcium salt in a 0.9% sodium chloride solution at a constant rate over four hours. Blood for serum calcium and phosphorus determinations is taken at 0, 2, 4, 8, and 24 hours with respect to the infusion period. Urine specimens for these determinations are taken during these periods and also during the day preceding and the day following the infusion. To know whether or not the 24-hour urine collections are complete, urinary creatinine should be determined for each 24-hour

period and any necessary corrections made in the excretion of phosphorus.

The diagnosis of hyperparathyroidism is highly suspected if (1) the rise in serum phosphorus is less than 1 mg. per 100 cc. during the period of hypercalcemia and if (2) the urinary excretion of phosphorus remains approximately the same as or is greater than the value obtained prior to the infusion of calcium. In normal persons hypercalcemia results in a rise in serum phosphorus over 1 mg. per 100 cc. and a significant reduction in the urinary excretion of phophorus.

The mechanism for these changes is uncertain. It has been postulated that hypercalcemia inhibits the secretion of the parathyroid hormone. This "shutting-off" reaction is considered to be less marked in patients with hyperparathyroidism than in normal persons—more hormone being produced, less "shutting off," and, consequently, relatively less renal reabsorption of phosphorus, resulting in lower serum phosphorus levels. However, Kyle, Schaaf, and Erdman [10] report that the elevation of serum phosphorus appeared to be related to the shift of inorganic phosphorus from the cellular compartment of the body rather than to phosphorus excretion.

In our experience, this test has been of definite help in certain problem patients. However, its reliability does not appear to be absolutely consistent, and frequently the difference between the normal and the hyperparathyroid patient is not sharply demarcated. The change in serum phosphorus levels may be consistent with the diagnosis of hyperparathyroidism, while the urinary excretion of phosphorus is not, and vice versa. The test is more tedious to perform than the phosphorus-reabsorption test. Chambers and associates [7] report a positive test from the standpoint of serum phosphorus changes in 7 out of 10 patients known to have hyperparathyroidism, but the urinary phosphorus changes were inconsistent. Schneider [11] and Kyle [12] report cases in which the test was of value in differentiating primary hyperparathyroidism from the milk-alkali (Burnett) syndrome.

Comment

These two tests have been of real help in the diagnosis of hyperparathyroidism. They are most valuable in those patients who have borderline levels of serum calcium and phosphorus and those with mild or early hyperparathyroidism. The phophorus-reabsorption test seems to be the more reliable and the simpler of the two tests used.

The phosphate-deprivation test has also been used as an aid in establishing or eliminating the diagnosis of hyperparathyroidism.[7] The patient is given a diet low in phosphorus but otherwise normal. This then is reported to accentuate the elevation of serum calcium and the decrease of serum phosphorus which occurs if hyperparathyroidism is present. We have not had experience with this test. It appears to be helpful, although the test must be carried out over a period of days. The magnification of blood changes of hyperparathyroidism may not always be very striking. It is of interest that, while the subject is on this diet, the renal tubular reabsorption of phosphorus increases in normal persons and approaches normal in patients with known hyperparathyroidism.

More experience with these tests is required for their complete evaluation. It is especially desirable that they be carried out in patients with other conditions which disturb calcium and phosphorus metabolism, and which may create problems in differential diagnosis.

Summary and Conclusions

1. The positive diagnosis of primary hyperparathyroidism is at times very difficult.

2. The tubular reabsorption-of-phosphorus, phosphate-deprivation, and intravenous calcium-load tests are of confirmatory value in the diagnosis of this condition.

3. The test for tubular reabsorption of phosphorus appears at this time to be the most definitive of these tests.

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Nonpenetrating Injuries of the Abdomen

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During 1956 it is estimated that 9,450,000 persons were injured in accidents in this country. The National Safety Council does not estimate the number of abdominal injuries. Among 2,000,000 disabling compensation injuries, 27% involved the trunk, and the abdominal injuries might make up one-third of these. The number would consist of both penetrating and nonpenetrating injuries. The former are commoner. This discussion is limited to the latter—nonpenetrating injuries of the abdomen. They are not large in number or in percentage of cases in the whole field of trauma. But the danger of visceral rupture or fatal hemorrhage makes this one of the most important injuries, and one in which the doctor's decision as to early correct diagnosis and early proper intervention may make the difference between life and death for the person concerned. The diagnosis is easier in penetrating than in nonpenetrating injuries. The mortality in nonpenetrating injuries of the abdomen is high—higher than in penetrating or perforating injuries. It is probably at least 20% to 30%. The duration and extent of morbidity is also increased over that for other abdominal injuries.

Nonpenetrating injuries are frequently a part of multiple injuries, with all the responsibility of doing first things first. Obvious, often severe trauma of other parts of the body too frequently masks, both to the patient and to the surgeon, the evidence or even the thought, of abdominal trauma. In any severe injury, particularly those incurred in automobile accidents, one must keep in mind always the possibility of abdominal injury, not only at the initial examination but throughout convalescence. If at the same time the patient is unconscious, the difficult nature of the decision is often tremendously increased.

No matter what other injuries a person may have, possible rupture of a hollow viscus requires that every effort be devoted to getting that person in condition for an abdominal operation at the earliest possible moment. Otherwise, tragedy is likely to result. Unconsciousness, a lung injury, or a badly contaminated open fracture is not a contraindication to abdominal exploration in the presence of a probable hollow-viscus injury or persisting intra-abdominal hemorrhage. The only contraindication is inability to counteract shock, and even here, when the expected response is not being obtained, operation may have to be done early as a possible life-saving measure, before irreversible shock sets in.

The cause of intra-abdominal injury may be a localized blow, e. g., in being thrown against a steering wheel, or a kick by an animal, or, more commonly, a crush, for example, between two trucks or a truck and a wall, or from falling rocks or falling walls. Explosion and blast injuries are rare causes. Probably the most frequent cause is the automobile accident. It must be remembered that the blow resulting in an intra-abdominal injury may be against the back or loin, as well as the abdomen. In some patients there seems to have been a "blow-out" of the small intestine from trapped air being suddenly compressed by an object.

Types of Nonpenetrating Injuries

The commonest injuries are to fixed organs—liver, kidneys, spleen, or urinary bladder. The most frequent sites of injuries to the intestinal tract are near points of fixation—the ligament of Treitz, the ileocecal

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junction, and the area adjacent to the attached portions of the large intestine. Rupture of the diaphragm with herniation of the stomach, large intestine, etc., into the chest is less common, but probably frequently unrecognized, and must always be kept in mind. It is usually on the left side and is frequently associated with injury to the spleen and left kidney. Avulsion of the mesentery of the small bowel or mesenteric thrombosis is frequent enough to be routinely searched for when the abdomen is open. Rupture of the pancreas and stomach is less common. Retroperitoneal rupture of the duodenum is rare, but so fatal, if unrecognized, that it deserves special mention. The patient may be asymptomatic for several hours, until there has been sufficient retroperitoneal leak. Testicular pain may be a symptom from irritation of the nerves retroperitoneally. The small intestine has been found wedged between two vertebrae, causing obstruction, and the literature contains reports of rupture of the gallbladder, hepatic ducts, Meckel's diverticulum, the bowel within a hernia, and the pregnant uterus. Chylous ascites has also been encountered after injury, as well as hemorrhage in the adrenals and rupture of major blood vessels, which it may be possible to repair.

Diagnosis

History.—The history, if obtainable, may be of considerable importance. Was the nature of violence localized or widespread? Was it over the lower ribs, so that liver, spleen, or kidneys may be involved? Is there injury to the vertebral column or pelvis so that retroperitoneal hemorrhage or bladder injury may be present? Was there a localized blow which might suddenly have compressed a distended loop of jejunum passing across the front of the vertebral bodies or might have torn mesenteric vessels? Has the person vomited, and did the vomitus contain blood? Has he voided since the accident? Is there a previous history of ulceration of the gastrointestinal tract, e. g., duodenal ulcer? A scarred area may be more subject to subcutaneous rupture. A carefully taken history, without delay of shock therapy, may give a valuable lead to required early therapy.

Shock.—When a person is seen in the first half-hour, he may not be in apparent shock. It is to be remembered that lowered blood pressure is a late, not an early, sign of shock. If the accident is of such a nature that shock might be expected, shock treatment should be started immediately, without waiting for the blood pressure to fall. The pulse is frequently slow for some time after injury. This should not give a false sense of security. Usually the person will be in shock when first seen and will require plasma expanders immediately, with properly checked blood as soon as it can be obtained. In cases of severe hemorrhage, intravenous blood administered under pressure, or furnished through all four extremities, or even intra-arterial transfusion may be the only means of correcting shock in that patient.

Examination.—At the first examination, abdominal tenderness at any site may be an important lead. At this time there is often no muscle rigidity, which later may mask tenderness. Localized rigidity of the abdominal wall from the retroperitoneal hematoma may be just as great as from an intraperitoneal injury, but usually does not come on as early. Rebound tenderness, if present, strongly suggests an intraperitoneal injury. Contusion of the abdominal wall should be looked for, since this, usually with increasing hemorrhage, may point to severe intra-abdominal injury. On the other hand, in more minor cases contusion of the abdominal wall only may produce apparently intraabdominal signs. Obliteration of liver dullness, shifting dullness, pain in the shoulder, and an abdomen quiet to auscultation are important if present. Frequent reexamination by the same surgeon is required.

Laboratory Studies.—The laboratory can aid in several ways. Of course, there are blood typing, Rh-factor determination, and cross matching. The blood count, hematocrit,

and blood volume, when obtainable within the critical period, are valuable. Urinalysis, particularly for red blood cells, is required. Serum amylase is usually elevated markedly and acutely with injury to the pancreas and diminishes gradually.

Roentgenologic Studies.—An x-ray film positive for free air in the peritoneal cavity is important. A negative finding does not rule out rupture of a hollow viscus. It is common routine to take this film in the upright position, and many of the patients cannot tolerate this position. Recently, several writers have reached the conclusion that "the left lateral decubitus position can be used to replace the vertical position of the abdomen and is of comparable accuracy in demonstrating free air in the peritoneal cavity." One should bear in mind that it may take several minutes for air to reach the highest point in the abdomen after change of position. X-ray exposures for bony injuries of the trunk, ribs, vertebrae, transverse processes, and pelvis are, of course, important, but we must not endanger life with the amount of x-ray studies we order. Have you ever watched the amount of exposure and moving of a patient that an x-ray technician seems to require for his work?

In rare instances the abdominal film fails to visualize any free air under the diaphragm, but one is struck with the clearness of outline of the right psoas muscle as compared with the left. Closer inspection may show areas of less density, which could be air bubbles. This is the picture of retroperitoneal rupture of the duodenum, and an absolute indication for early operation.

With fracture of the lower ribs, upper lumbar transverse processes, or bodies of the upper lumbar vertebrae, kidney injury must be considered. With fracture of the pelvis one must look for bladder or urethral injury. In either instance a portable x-ray examination is useful and can be done at the bedside, using a Lysholm grid over a standard cassette in a cassette tunnel, with little disturbance to a badly injured person. The intravenous pyelogram may be of considerable help in determining a kidney injury. This should be done within the first two hours. Further films at one-half and one hour or longer may give an excellent cystogram or show extravasation from the bladder or posterior urethra.

If there is a possibility of bladder or urethral injury, an attempt should be made to pass a 16 F. soft-rubber catheter. Obtaining clear urine does not necessarily mean that the bladder and urethra are intact. Gross blood may mean severe contusion without rupture. Failure to pass a catheter may be due to a stricture of the urethra or an obstructing prostate, and not to a ruptured urethra. When passed, the catheter is clamped, and intravenous pyelography done. If this cystogram proves unsatisfactory, the catheter is unclamped and contrast material is injected retrograde. Instilling a measured amount of fluid and noting whether all is returned, as a test of lower tract rupture, is mentioned, only to be condemned.

Abdominal Tap.—We believe that this may be a valuable aid in determining the need for exploration in the questionable case. It means nothing, one way or the other, if found negative. But if blood, air, or turbid fluid is obtained, the tap is diagnostic. A spinal needle, 18-gauge or larger, with the trocar in place, is inserted 5 cm. to the right and 5 cm. below the umbilicus unless there is a definite lead to an involved area. This needle must be absolutely dry, and the syringe and the barrel must fit tightly. Even though nothing seems to be withdrawn, the possible contents of the needle should be expressed onto a clean slide for immediate examination for blood or other cells.

Early Care

Time of Operation.—Definite evidence of hollow-viscus injury requires operation at the earliest time the patient can be gotten in shape to stand it. Until it can be carried out, there is no situation in medicine demanding more careful and frequent

observation and balanced judgment. The responsibility should be on a senior attending surgeon, not a junior resident, and the patient should be seen frequently by the same surgeon. Often little is shown at first; for example, not much peritoneal irritation, no severe general reaction, no disturbance of the blood picture. But if the signs progress, that is, if the pulse rate increases, the hematocrit drops, peritoneal irritation increases or spreads, or vomiting or distention develops, we must consider any of these as greater evidence of intra-abdominal injury and cause for reevaluation of the need of exploring at that moment. After all methods of examination indicated have been carried out promptly and the surgeon has examined the patient several times, there will be some instances in which the findings are still indefinite but cause anxiety. Then the most conservative procedure for an experienced surgeon may be to do a laparotomy on the grounds of suspicion only.

Preliminary Measures.—A thoracoabdominal injury requires stabilization of the intrathoracic physiology before proceeding. The sucking chest must be sealed to relieve mediastinal flutter. A major area of paradoxical respiration may require traction. A tension pneumothorax will need immediate needle aspiration. Pneumothorax or atelectasis may have shifted the mediastinum sufficiently so that an abdominal procedure is likely to be lethal unless the cardiorespiratory disturbances have been relieved first.

Adequate blood replacement must be at hand, for release of abdominal pressure by incision may result in immediate extreme hemorrhage, particularly in case of liver injury. Many of these persons may have had a meal shortly before. Washing out the stomach before anesthesia may be a life-saving procedure.

Anesthesia.—Intratracheal anesthesia is probably the safest. Ether anesthesia comes next, and in the bad risk is preferably administered by open method. Spinal anesthesia is indicated rarely, since one can never be certain what part of the abdomen may have to be entered or how prolonged a procedure may be required. Local anesthesia rarely has any place. A free airway and adequate oxygenation are most important.

Incision.—This must be a generous one and give easy access. These patients are not cases to stand heroic pulling on retractors by assistants or constant shifting of place and direction of retractors. Careful evaluation of all the symptoms and signs is required to place the incision where the most important lesion is probably located. The stomach and intestine may need to be examined for their entire length; consequently, the exposure must ultimately be large enough so that all fixed points may be brought into view.

Exploration and Repair.—Suction must be running on opening the abdomen. The liver is the organ damaged most commonly in nonpenetrating injuries. Frequently the hand makes the best pack until the abdominal cavity can be cleared of blood. The hepatic artery and portal vein may be largely controlled by getting them between thumb and fingers in the anterior margin of the foramen of Winslow. The thumb and finger may control a splenic pedicle or mesenteric vessels until the field has been cleared enough by suction, and packed, so that one can judge the source of major bleeding. Oxidized cellulose U. S. P. (Oxycel), absorbable gelatin sponge U. S. P. (Gelfoam), and the like are life-saving packings in the liver and may often need to be sutured into the laceration to prevent later displacements. It is to be remembered that the lead to diagnosis of laceration of the liver is often fracture of lower right ribs. The right lobe is injured five times as frequently as the left, and twice as frequently on the convex as on the concave surface. Diseases of the liver may increase its friability so that it is predisposed to injury from minor violence. Bleeding from the liver may be slow or intermittent following clotting. An intrahepatic hematoma may lead to hepatic abscess months later. The lacerations are often multiple or radial from a central area. One should not forget that continuing hemor-

rhage from a site not located is often from the dome of the liver. On recognition, the incision should be extended immediately so that further procedure will be thoracoabdominal, giving ready access to the dome. Occasionally a subcapsular hemorrhage of the liver is seen. One cannot be certain of the extent of this from the external appearance. If left alone, the capsule may rupture later, with a fatal hemorrhage. It should be opened, the clots evacuated, and packing, or suturing or both resorted to. Partial or complete resection of a lobe is rarely indicated, and then only in experienced hands. Commonly the condition of the patient will not allow this procedure. Adequate multiple drains should always be inserted in the region of the liver laceration. Recurrent hemorrhage may then be recognized, and bile peritonitis is less likely to occur.

In spite of repeated warnings, rupture of the spleen is frequently missed. Children injured when a sled collides with an object are sent home too often because they seem symptomless. Fracture of lower left ribs anteriorly or laterally usually requires hospital admission for prolonged careful observation. One must not forget that sudden fatal hemorrhage has occurred as late as three weeks after injury, or even longer. Splenic rupture is often masked by the presence of other injuries which may seem of paramount importance. A tumor mass in the left upper quadrant, with any possible history of recent injury, must make one suspect an injury to the spleen. The spleen when found bleeding, however minimally, should not be packed or sutured, but always removed. The spleen which has a subcapsular hematoma of even moderate extent should also be excised. The capsule does not stretch well, and its later rupture is too common, and may result in death before being recognized or cared for.

Perforation of the stomach is uncommon, usually resulting from heavy pressure against a filled stomach. The viscus may be bruised or lacerated by being crushed against the vertebral column or ruptured by the explosive hydraulic effects of compression. The fuller the stomach the more vulnerable to compression. The commonest site is in the lesser curvature. Suture is the treatment, when the rupture is found. In exploring the right upper abdomen, one may rarely see a grayish-black swelling beneath the posterior peritoneum or in the mesentery of the ascending colon and palpation may give crepitation. This indicates a retroperitoneal rupture of the duodenum with leakage, and death will result if it is not visualized and repaired. While this lesion is rare, its x-ray signs, as described previously, and the clinical signs noted above, if kept in mind, may mean the saving of a life. Tears of the intestine are most frequent near fixed points, but may occur anywhere that severe pressure is exerted against the abdominal wall when a loop of distended intestine lies over the vertebral column. If a hollow viscus is filled or if there has been preexisting pathology, the danger of rupture is increased. Injury to the jejunum is commonest, but I have seen tears several times in the ileum. This lesion must be remembered in fractures of the pelvis. The intestine is involved in 1 out of 10 subcutaneous injuries of the abdomen. There may also be a laceration or an avulsion of the mesentery without direct damage to the intestine. These lesions are usually closer to the free margin than to the root. Mesenteric thrombosis may follow, with secondary infarction of the affected bowel and gangrene. Contusion and hematoma of the bowel, after hours or days, may lead to perforation with peritonitis. After such injuries pulmonary fat embolism is a common cause of death. Wider tearing is commoner in the large intestine and may require exteriorization, rather than resection. This is not true in the small intestine.

Injuries to the rectum require colostomy plus exposure and suture, if possible, on account of the probable retroperitoneal infection.

With a diagnosis of retroperitoneal hematoma, as distinct from an intraperitoneal

lesion, no exploration should be done. When one is encountered as a part of exploration, it should be left alone unless the signs of retroperitoneal duodenal rupture, as mentioned above, are present.

When the pancreas is damaged by external violence, it is usually a small part of an extensive injury. If interstitial bleeding results in the formation of a hematoma, subsequent liquefaction of the mass of blood may lead to the formation of a cyst. Traumatic or pseudocysts have no distinct lining membrane. When a laceration of the pancreas occurs, hemorrhage is usually profuse into or behind the peritoneal cavity. This is often followed by acute pancreatitis with fat necrosis and chemical peritonitis. The serum amylase is usually markedly elevated. An injury to the pancreas may be found at operation. This is usually not of a serious nature, but when found it should be sutured as a protection against further bleeding and leakage of pancreatic secretions. It is advisable to place a drain down to the site of injury. One should not forget that pancreatic pseudocysts may form later.

In injuries of the kidney immediate operation is rarely required. Rupture of the bladder requires immediate operation almost as urgently as that of any other hollow viscus. If the perforation is not found readily, however, or if it is not at a site which can be closed readily, one should be satisfied with suprapubic cystotomy.

Rupture of the diaphragm occurs more frequently than is recognized. The commonest preoperative physical sign is the x-ray finding, with a tube in the stomach for decompression, that the stomach is in the chest. Without a tube an elevated gastric bubble may be seen and a swallow of barium should be given. When the rupture is recognized before operation, a thoracic or thoracoabdominal approach is usually preferred. Whenever even a small rupture in the diaphragm is found, it should be sutured, to prevent later difficulty. It should be remembered that unexplained chest symptoms or signs may be from a ruptured diaphragm, with abdominal viscera in the thoracic cavity, causing pressure.

Closure of the abdominal incision should be meticulous. Vomiting, distention, and restlessness are frequent complications, and disruption of the wound adds a tremendous hazard. Therefore reinforcement with wire closure is often advisable.

Comment

Have we planned and organized hospital care so that these infrequent, but often lethal, injuries are given the best attention of which we are capable?

Many of our hospitals are excellently organized for resident training between 9 a.m. and 5 p. m., but provide little supervision for the resident staff during the remainder of the day and through the night. This is frequently the period of the severest automobile accidents, and the time that we may need to give most advice and assistance to our resident staffs, when indicated. Do we really have resident training if we let them work out their own salvation on these severely injured patients, who may die if first things are not done first?

In the emergency room in your hospital, who examines an injured person first? May it be an intern at the most junior level, who has never seen a traumatic case before, or an indifferently trained foreign physician with language difficulty? Have you prepared and posted a directive which will give these junior men an idea of what instances require the immediate notification of a surgical resident? In the middle of the night, will this be a resident who has been instructed how to recognize the signs of intra-abdominal injury and to call a member of the attending staff immediately, and not wait until the next morning? Will the attending staff member notified be at the most junior level or a senior man with experience? Can you decide over the telephone, from the description given, that the patient needs an immediate urological consultation rather than observation by you? Do you know from personal inspection what goes on in your

emergency room in the middle of the night, or do you stay away, because of a subconscious fear of what you might see?

There is little doubt in my mind that the weakest link in the chain of hospital care is the emergency-room attention to the injured in most hospitals in this country. Suppose that you were injured in an automobile accident in your own city tonight, that you were rendered unconscious and that your identity was not recognized, that you were brought to the emergency room in your own hospital still unconscious and unidentified with multiple injuries, including possible abdominal trauma, do you believe that your intern and resident staff have been so trained that you would be handled in a way which would probably save your life? If your answer is "no" or questionable, how can you expect adequate handling in some other town where the same sort of accident might happen to you? As a matter of fact, I believe that the smaller the community with a hospital to which such an accident is brought, the greater the likelihood of competent care. How long is it since you have had a medical-board survey of your emergency room?

Internships and residencies in hospitals are for practical training, not for an economic aid to hospital finances. The training is necessary because it is no longer possible to get it in a medical school with the myriad of basic principles which must be gone over. Probably 50% of the practice of most physicians starting in will be in the traumatic field. Are you seeing to it that from their training in your hospital they will be prepared to do justice to these injured?

Summary

Nonpenetrating injuries of the abdomen present a challenge to any surgeon. Multiple injuries frequently mask the abdominal one almost completely. Shock and blood loss are likely to be extreme and must be combated promptly and effectively. Meanwhile, every method of diagnosis must be used at the moment when it will not harm the patient further. Time and laboratory reports will not settle this problem for the surgeon. He must depend on his own hands and brain. The decision as to time of operation when indicated may mean the balance between life and death. Indecision may lose for the patient the only chance he has. The surgeon who meets these cases infrequently must appreciate the great responsibility incurred.

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Identification of Cause of Obscure Massive Upper Gastrointestinal Hemorrhage During Operation

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Introduction

Many important contributions have been made to knowledge of massive upper gastrointestinal hemorrhage in recent years. The importance of coordination of medical and surgical action and planning by a "team" approach has been stressed by us and by others.[1,2] The value of establishing a preoperative diagnosis is self-evident.[3] Major contributions in this area have been made by Hampton,[4] who reported limited x-ray studies during or shortly after hemorrhage. The study can be helpful, as has been shown by Ritvo, Cotter, and Zamcheck,[5] who report the safety and accuracy of complete upper gastrointestinal tract roentgenograms. The importance of liver disease as a complicating factor in diagnosis and prognosis is evident from the studies of Smythe and others.[6] The sulfobromophthalein (Bromsulphalein) test has proved particularly useful in screening patients with significant liver disease [7] and has been used in connection with esophagoscopy [8,9] as a means of identifying bleeding esophageal varices. Palmer [10] has shown that gastroscopy is helpful in experienced hands. Some factors which assist in determining which patient should be treated surgically are indicated in Figure 1.

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Despite these advances, massive upper gastrointestinal hemorrhage still poses a particularly trying problem for both physician and surgeon. Even with careful history and physical examination, early and complete x-ray studies, liver function studies, and endoscopy, the site of bleeding cannot be identified preoperatively in many cases. Moreover, because of the gravity of the hemorrhage in some cases, it is not always possible to employ all of the diagnostic aids preoperatively. Figure 2 shows the majority of causes of acute massive upper gastrointestinal hemorrhage. It can be seen readily that many of these lesions can be identified only by direct inspection in the operating room. A typical example of this type is shown in Figure 3; an acute, massively bleeding, but superficial gastric ulcer.

It is to a consideration of this problem, namely, what to do with the patient in whom the site of bleeding cannot be recognized, that this paper is directed. At the outset, one safely assumes *upper* gastrointestinal tract origin of bleeding in the patient whose symptoms clearly point to this location, whose x-ray is diagnostic, who has demonstrated hematemesis or from whose gastric drainage blood is recovered, or who is found to have blood in the stomach or upper small bowel at operation.

Several approaches to this problem have been recommended in the past. In the first instance it has been thought that when the bleeding lesion could not be identified preoperatively, surgery should be postponed in the hope that bleeding would subside spontaneously and that more accurate diagnostic studies could be done at an interval period.

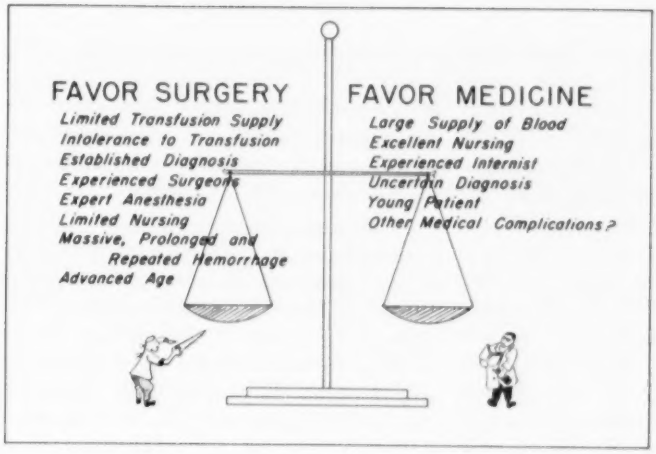

Fig. 1.—Factors which favor medicine or surgery.

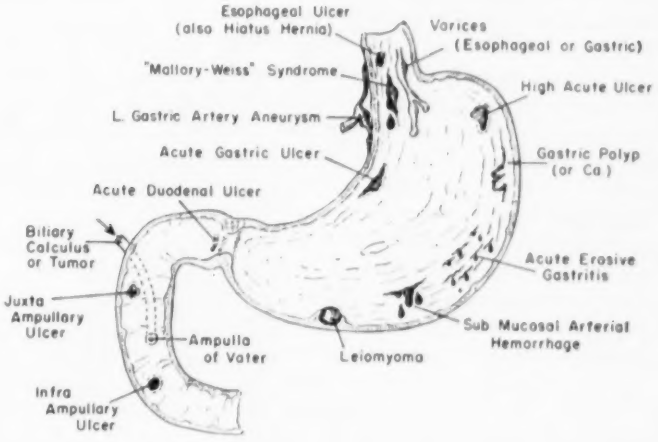

Fig. 2.—Upper gastrointestinal tract lesions which give rise to most massive hemorrhages.

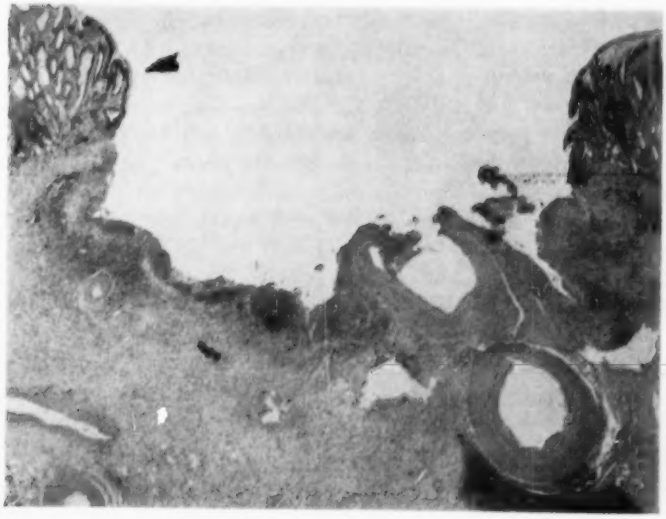

Fig. 3.—Acute, massively bleeding gastric ulcer. This lesion cannot be seen with x-rays and will not be palpable in the intact stomach.

Further experience with this problem, particularly with debilitated patients in a city hospital population, has convinced us that the mortality in the obscure bleeder is particularly high and that if bleeding is massive, surgery should not be postponed. A second approach has been recommended by Stewart and associates,[11] Thieme,[12] Cooper and Ferguson,[13] and others, namely, prompt surgery with "blind" gastrectomy. Although we realize that "blind" gastrectomy may be required because the lesion cannot be identified at operation, we feel that over emphasis of this approach leads to failure to identify lesions which will not be removed by gastrectomy or the performance of this operation for lesions which might not have required it. This is particularly true of the surgeon who has not had an extensive experience with the obscure bleeding lesion and who under the stress of an emergency operation may overlook a demonstrable site of bleeding because he does not employ a systematic examination during laparotomy and fails to appreciate the nature and variety of pathological lesions concerned. Spencer and Maloney[14] recently have pointed out the value of gastrotomy in this connection, an experience which is confirmed by our own.

It is our purpose in this communication to examine the problem of surgical intervention for massive upper gastrointestinal hemorrhage with regard to the question of diagnosis during operation and to appraise the importance and feasibility of achieving this objective. The operative technique employed has been presented elsewhere[15] and will be recounted here only in outline form.

1. As the abdomen is opened, it is to be noted whether or not blood clots in the wound. Clotting defects may develop as a result of multiple transfusions, and it is important to determine the state of coagulability of the blood as the wound is being made. As the abdomen is opened, one should look for evidence of extensive portal collateral circulation in the abdominal wall and falciform ligament. The presence of ascites and peritoneal seeding and the appearance and consistency of the liver can be determined in a matter of seconds. Similarly, before proceeding with exploration of the upper gastrointestinal tract, one should note the presence of other gross visceral disease, such as cholecystitis or hepatitis.

2. The course of the operation is affected to some extent by whether or not the patient has had hematemesis and/or whether blood can be demonstrated in the stomach at the time of operation. Either finding directs attention to the upper gastrointestinal tract and provides the surgeon with assurance that the bleeding point is above the ligament of Treitz.

3. The upper gastrointestinal tract should be palpated and inspected. No clamps of any kind should be placed on the stomach. The stomach is drawn into the wound gently, inspected, and palpated against the pancreas and bimanually. The esophageal hiatus and the spleen are palpated. The duodenum is next inspected and palpated, noting local edema, adhesions, scarring along vascular paths, and punctate hemorrhages. Punctate hemorrhage after palpation suggests an underlying ulcer provided no clamps have been used. Assuming that no lesion has been found, the lesser sac is opened and the stomach palpated bimanually again. The posterior aspect of the stomach is inspected for adhesions or inflammatory changes. The duodenum should now be mobilized for better inspection and palpation. This step is usually useful regardless of the lesion found, since it greatly facilitates gastrectomy.

4. If no lesion has been found, there has been no hematemesis, and there is no evidence of blood in the stomach or duodenum, one should focus attention upon the upper part of the small bowel, especially seeking diverticula or small vascular tumors. Transillumination of the upper small bowel is quickly and easily performed for a distance of 2 or 3 ft.

5. If no lesion is found, gastrotomy should now be performed. Careful technique is essential. Trauma to the gastric mucosa by suction, sponges, instruments,

TABLE 1.—*Sex, Average Age, and Differential Mortality Among 100 Surgically Treated Patients*

Sex	No. of Patients	Average Age, Yr.	Deaths, %
Male	87	57.7	26
Female	13	58.9	31
(Died)	(26)	(66.5)	
Total	100	57.9	26

and coarse palpation must be avoided. The stomach is opened through an initially small incision, and clamps are applied to the gastric wall so as completely to control bleeding as the incision is extended. An incision at least 15 cm. in length is necessary for adequate inspection. The presence of blood in the stomach and the condition of the gastric mucosa, especially along the lesser curvature and on the posterior gastric wall, are noted. The upper portion of the fundus may be inverted into the operative incision. After thorough inspection, the interior of the stomach and of the lower esophagus is palpated.

6. If no lesion is found, the gastric outlet should be inspected carefully and the duodenal cap palpated through the pylorus. About two-thirds of the duodenal cap may be everted back through the pylorus for inspection. It may now be necessary to extend the gastrotomy incision across the

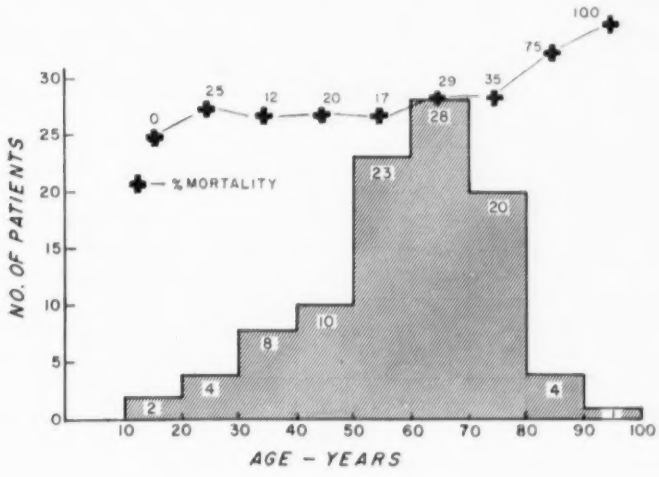

Fig. 4.—Age and mortality.

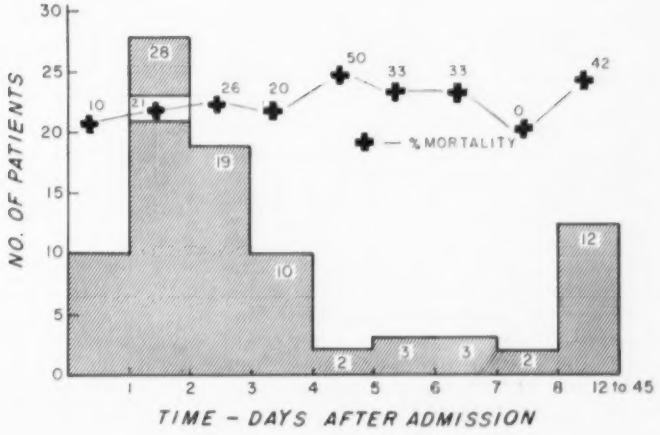

Fig. 5.—Time of operation and mortality.

pylorus and inspect the duodenum directly. The gastrotomy incision will be closed with a running suture prior to gastrectomy if that is elected.

7. If no lesion has been found but blood wells back from the distal part of the duodenum, a bleeding point in the second or third portion of the duodenum is suggested. If no lesion can be established on palpation of the mobilized duodenum, before opening its second portion, a small incision may be made in the jejunum just beyond the ligament of Treitz and the entire duodenal loop irrigated with saline. Continued passage of blood in the irrigating fluid indicates bleeding proximal to the ligament of Treitz and distal to the first portion of the duodenum. Under these circumstances it is now permissible to carry the duodenotomy incision distally.

8. If an upper gastrointestinal tract origin of bleeding is not certain, the small bowel and colon may be examined by palpation and transillumination, with special attention to the terminal ileum and the cecum. Suspicious areas may be checked through stab wounds.

With use of these methods, our need for "blind" gastrectomy has been markedly reduced.

Material

One hundred consecutive cases of severe acute upper gastrointestinal hemorrhage treated by surgical intervention on an emergency basis on the Fifth (Harvard) Surgical Service of the Boston City Hospital have been studied. Operations were all done during or immediately after bleeding episodes which were regarded as life-endangering if surgery were not undertaken.

The data on the patients are summarized in Table 1. It is noteworthy that among the 87 males and 13 females there was a high gross hospital mortality (26%) and that 53 of the patients were more than 60 years of age (Fig. 4). These patients received between 3 and 13 units of blood to replace losses before and during surgery, with an average of 3850 ml. in transfusion. These massive blood requirements reflect the severity of the hemorrhages. The urgent character of surgery is shown in Figure 5; 10 patients were operated on within 24 hours of admission; 28 more within the next day, and a total of 57 operations within 1 more day. Twenty patients who were hospitalized for more than five days prior to surgery all exhibited recrudescent (secondary) bleeding.

TABLE 2.—*Final Diagnosis of Bleeding Lesions*

Duodenal ulcer	
Acute	17
Chronic	38
Gastric ulcer	
Acute	15
Chronic	17
Gastritis	6
Gastric carcinoma	2
Unknown	2
Esophageal varices	1
Marginal ulcer	1
Small bowel aneurysm	1
Total	100

Because of the desperate condition of the patients, x-ray study was not attempted or could not be completed satisfactorily in 38 cases. In six instances x-ray examination had been done previously, and it was not thought necessary to repeat it. In 49 cases the x-ray examinations were done and were helpful in localizing the lesion and its nature. In eight cases the x-ray was negative, and in 4 it led to an incorrect diagnosis.

Results of Surgery

The mortality among these patients has been described. Table 2 lists the diagnoses derived from the operators' descriptions and the pathologists' studies. There were 31 examples of acute peptic, gastric, and duodenal ulcers. In 26 of these the diagnosis was not firmly established until gastrotomy was performed. These lesions presented a major problem in diagnosis at the time of surgery. Gastritis (six cases), a marginal ulcer, and a small-bowel aneurysm also required direct inspection for their identification. In one case of known cirrhosis a diagnosis of bleeding esophageal varices was correctly made at laparotomy after negative esophagoscopy. Subsequent esophagoscopy and thoracotomy confirmed the presence of bleeding varices. In only two cases was no diagnosis established at operation; in both a "blind" gastrectomy was done for what the surgeon interpreted as minimal evidence of duodenal ulcer. In both cases the pathological examination disclosed no lesion. In both instances the patient did well and there has been no further bleeding. The operations on this group of 100 pa-

TABLE 3.—*Operations*

For diagnosis	
Gastrotomy	40
Esophagoscopy	4
Definitive operation	
Gastrectomy	96
(7 "blind")	
Exploratory gastrotomy only	1
(later varices ligation)	
Local lesion excised	3
	100
Associated procedures	
Splenectomy	2
(for operative injury)	
Common bile duct intubation	2
Vagotomy	2
Varices ligation	1
(postgastrotomy—1 day)	
Ulcer suture locally	1
(pregastrectomy—1 day)	
Ulcer suture locally in duodenal stump	1
(postgastrectomy—1 day)	

tients are indicated in Table 3. Partial or subtotal gastrectomy was performed 95 times and total gastrectomy once. The four other definitive procedures included excision of a small-bowel aneurysm following gastrotomy in one patient. One acute, bleeding gastric ulcer over a traumatic pancreatic pseudocyst was excised locally. An acute gastric ulcer was excised locally in a patient with severe pulmonary tuberculosis. In the fourth case, bleeding esophageal varices were recognized at the time of gastrotomy, and subsequent transthoracic ligation of the varices was performed.

Associated procedures included two vagus-nerve sections, and one transverse colostomy, done on account of an associated carcinoma of the colon. Splenectomy was necessitated twice by operative injury to the spleen; common bile duct intubation was done for security on two occasions. One patient, whose ulcer defied excision, required transfixion suturing on the day after gastrectomy, when secondary hemorrhage through a tube duodenostomy became evident. Another patient required emergency gastrectomy for recurrent hemorrhage one day after a bleeding duodenal ulcer had been sutured locally.

Gastrotomy and "Blind" Gastrectomy

Gastrotomy was employed in 40 of these 100 operations. Occasionally, duodenotomy, duodenal (to jejunal) irrigation, small-bowel transillumination, careful inspection of gastric remnants, and inspection of the duodenal interior before closure of the stump were combined with gastrotomy in the effort to locate bleeding points. A correct diagnosis was made at operation in 37 of these 40 cases.

In the three cases in which diagnosis was not revealed by gastrotomy, and in four other cases, gastrectomy was done blindly. In one of these seven cases a small chronic duodenal ulcer was removed, with a good result; in the second multiple, small gastric erosions were seen in the resected specimen, but the patient died of medical complications; in the third the specimen showed a minimal gastritis, bleeding continued, and the patient died. The fourth and fifth cases yielded entirely negative specimens, but there was no further bleeding and results were good for both patients. The sixth "blind" gastrectomy was performed for what was diagnosed as gastritis by the pathologist, but postoperative bleeding continued and was fatal, and autopsy showed multiple gastric ulcers in the gastric remnant. A gastrotomy could have prevented this death. In the last case gastritis was diagnosed by the pathologist, and the patient required seven transfusions for continued bleeding after operation, but a good result was finally achieved. These cases of "blind" gastrectomy are summarized in Table 4. Three of the seven patients so treated died, a 42% mortality.

TABLE 4.—*Results of "Blind" Gastrectomy*

Case No.	Pathology Report	Comment	Result
1	Chronic duodenal ulcer	Gastrotomy neg.	Good
2	Multiple, acute gastric ulcers	Gastrotomy neg.; myocardial infaret	Died
3	Minimal gastritis	Gastrotomy neg.; bleeding continued; myocardial infaret	Died
4	Negative stomach	Duodenal palpation misinterpreted by operator	Good
5	Negative stomach	Minimal signs of duodenal inflammation seen at operation	Good
6	Gastritis	Bleeding continued; autopsy showed gastric ulcers and cirrhosis	Died
7	Minimal gastritis	3500 ml. of blood required for postgastrectomy bleeding	Good

TABLE 5.—*Diagnoses Established by Gastrotomy*

Diagnosis	No.
Acute gastric ulcer	16
Chronic gastric ulcer	4
Acute duodenal ulcer	10
Chronic duodenal ulcer	1
Gastritis	3
Esophageal varices	1
Small bowel aneurysm	1
Marginal (jejunal) ulcer	1
No diagnosis	3
Gastrotomy—Total	40

The 37 diagnoses made at gastrotomy are listed in Table 5. Fourteen of these patients died, a 35% mortality. They represent the most serious and troublesome group. Their surgery could not be accomplished as expeditiously as in cases in which the diagnosis is obvious to the operator. The benefits of gastrotomy and other measures used to diagnose these bleeding lesions with maximum certainty may be summarized as follows:

1. Maximum security with regard to the appropriateness of a given operation was achieved when the diagnosis was verified.

2. Three lesions in this series could be treated by local removal, with good results.

(*a*) An acute bleeding gastric ulcer over a post-traumatic pancreatic pseudocyst was excised and cystogastrostomy performed.

(*b*) A bleeding arterial aneurysm in a diverticulum of the upper jejunum was excised locally after gastrotomy had shown gastritis without local bleeding.

(*c*) An acute bleeding gastric ulcer was excised locally in a patient with far-advanced, bilateral pulmonary tuberculosis.

3. There were no negative pathological reports rendered on specimens submitted for this group of 40 patients.

4. Five patients had bleeding lesions excised by gastrectomy which would have been missed by any "blind" resection.

5. A large amount of blood loss was obviated by early control of bleeding points locally.

6. An error of esophagoscopic diagnosis was corrected.

These three cases are presented briefly to illustrate the importance of exact diagnosis during surgery.

Illustrative Cases

CASE 1.—A 70-year-old white man was admitted because of hematemesis two hours before. Hematemesis and melena five years previously had been treated conservatively. X-rays had shown small-bowel diverticula.

The admission hematocrit was 18%; the patient was pale, cold, and sweating, with pulse 108 and blood pressure 110/70. He had been a known hypertensive. He required 1000 ml. of blood daily to maintain a stable circulation but continued to bleed. On the third day hematemesis recurred. The hematocrit was only 14%, and surgery was elected after rapid blood replacement. At operation, gastrotomy showed minimal gastritis but no bleeding lesion, and careful reexamination of the upper small bowel disclosed a very small submucosal aneurysm in a jejunal diverticulum close to the ligament of Treitz.

Comment.—Extensive "blind" gastrectomy would have failed to remove this bleeding lesion.

CASE 2.—A 47-year-old white man was transferred from a tuberculosis sanatorium because of melena, weakness, and anemia, of two days' duration. He had been under treatment for bilateral pulmonary cavitary disease and tuberculous laryngitis with streptomycin and aminosalicylic acid U. S. P. for 18 months.

The hematocrit was 12% on admission, and there was persistent hematemesis. Infusion of 2500 ml. of blood was given during the first eight hours, and surgery was elected when hematemesis continued. Operation revealed an acute bleeding gastric ulcer, which was seen on gastrotomy and was resected locally. The patient was returned to the sanatorium after a convalescence prolonged to 73 days by wound infection and poor healing.

Comment.—Gastrotomy permitted a local resection of the bleeding lesion, a life-saving step for this patient. It averted a gastrectomy and its attendant adverse effects in a patient with advanced tuberculosis. It was felt that aminosalicylic acid therapy should be discontinued and that this omission, with ulcer management, would protect the patient from recurrence.

CASE 3.—A 15-year-old white school boy, previously entirely asymptomatic, was admitted because

of bright-red bleeding from the rectum, of one day's duration.

Hematocrit was 28% on admission. An infusion of 4000 ml. of blood was given during the next 30 hours, and red, bloody stools persisted, along with repeated episodes of shock. The hematocrit fell to below 20% despite transfusion. An emergency G. I. series and barium enema studies were negative. At operation, gastrotomy showed blood welling back from the second portion of the duodenum. Irrigation from the pylorus to an incision in the jejunum just beyond the ligament of Treitz confirmed the presence of active, fresh bleeding. Duodenum was opened progressively until finally a deep, acute, juxta-ampullary bleeding ulcer was revealed. Partial gastrectomy and duodenectomy were performed, and the patient was discharged 10 days later, after an uneventful course. Nutrition has been adequate since operation (three years later), and there has been no recurrence.

Comment.—Tragedy was averted when this lesion was identified and removed; it is likely that the massive hemorrhage from the exposed pancreatoduodenal artery would have persisted despite "blind" gastrectomy.

Summary and Conclusions

Despite many advances, severe, acute massive upper gastrointestinal bleeding still poses a serious problem for both physician and surgeon. This is especially true when preoperative study fails to reveal the cause of bleeding.

In almost every case a correct diagnosis can be established at operation if a systematic, careful exploration is performed, and this procedure has reduced the need for "blind" gastrectomy.

When a preoperative diagnosis cannot be made and serious hemorrhage continues, operation should not be delayed.

Experience with 100 surgical cases is presented, and a systematic method for exploration of the bleeding patient is described.

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Closed Reduction of Common Shoulder and Elbow Dislocations Without Anesthesia

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The purpose of this paper is to emphasize the simple fact that the majority of common shoulder and elbow dislocations can be reduced without anesthesia, without increased pain or trauma to the patient.

Of the 28 dislocations of the shoulder and of the 20 dislocations of the elbow we have encountered at the U. S. Army Hospital, U. S. Military Academy, since June, 1954, all were reduced without anesthesia and without significant increased pain or trauma. Since the initial preparation of this paper, two additional dislocations of the shoulder and three additional dislocations of the elbow have been encountered.

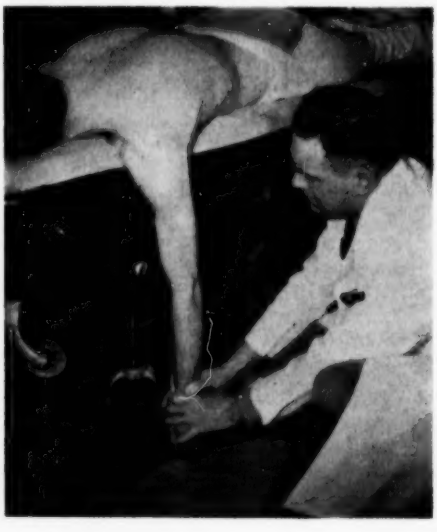
Shoulder Dislocations

One of three methods (none completely original with me) was used in reducing the dislocated shoulders in this series. The method most frequently used was one similar to the Milch technique, advocated by Lacey and Crawford.[1] The second method is Boehler's method, which is actually a self-reduction method. The third method, and one used in only two instances, when the first two methods failed, is that of Bennett. The chief advantages of any of these methods are those inherent in avoiding the hazards of general anesthesia and reducing the painful interval of time between dislocation and reduction. Lest this statement be misconstrued, it should be pointed out that the actual manipulation time may be relatively long and that these methods are not suited to the impatient "quick-pull" artist. For any of these methods a sedative is desirable, but not necessary. Indeed, I do not routinely administer a sedative, since reduction is usually accomplished before the sedative could possibly be effective.

I have not used an anesthetic to effect a reduction of a shoulder dislocation since 1951, but frequent changes of military assignments has precluded documentation of cases to prepare an authentic report. Prior to 1954, I reduced shoulder dislocations most commonly by the Boehler method, but since June of 1954, the method similar to the Milch technique has been used almost exclusively.

This most commonly used (the Milch) method is one in which the patient is placed in a prone position on a high table or litter with the affected extremity hanging freely over the side of the table (Fig. 1). Occa-

Fig. 1.—Reduction of shoulder with dependent traction. Note that scapular portion of shoulder is well over the edge of the table, resulting in traction in abduction.



From U. S. Army Hospital, U. S. Military Academy, West Point, N. Y.

sionally after only a few minutes in this position the weight of the arm alone is sufficient force to reduce the shoulder. However, traction is usually required. The operator sits on a low footstool or on the floor, grasps the patient's wrist gently but firmly, and exerts steady downward traction. If reduction is not effected by five to six minutes of sustained traction, the reduction may be aided by flexing the elbow about 25 degrees and slightly rotating the arm externally with traction maintained. While traction is being applied, the patient is continually advised to tell the operator whether significant increase in pain is encountered. If so, the traction is diminished, inasmuch as painful traction results in increased spasm and failure. I believe this method to be the simplest and least traumatic of those methods wherein the reduction is effected by the surgeon.

Boehler's method (so-called by me because it was learned from Prof. Lorenz Boehler) is one wherein the patient is placed on a stool, preferably a swivel-top stool, and helped to grasp the leg of a fixed or heavy piece of furniture with the hand of the affected extremity at waist height (Fig. 2). After being instructed to take all the time necessary, the patient is asked to maintain a firm grip on the leg of the piece of furniture and start rotating his body on the swivel-top stool in the opposite direction, so that he actually effects external rotation of the arm. When it begins to be painful, the patient is coached to let up on the rotation a little until he gets used to that position, and is then directed to resume rotation. Somewhere between 70 and 110 degrees of external rotation spontaneous reduction results. Here, again, haste must be avoided, or failure will result. This method is not suitable for the emotionally unstable person or a patient with a labile blood pressure and a tendency toward fainting. It is an excellent method to teach patients with recurrent dislocations, in order that they may reduce their shoulder dislocations when help is not available. It is an excellent method when patients are unable to lie on the abdomen, as in the case of advanced pregnancy. Indeed, Ramsey [2] had a pregnant patient with dislocation of the shoulder and reduction was accomplished by this technique.

The third method, used in only two cases in this group, is Bennett's (Fig. 3). In this method the patient is recumbent on a litter or table. The operator flexes the elbow of

Fig. 2.—Self-reduction of shoulder. Rotation of patient on stool effects external rotation and abduction of humerus.

Fig. 3.—Reduction of shoulder by traction in abduction by using body weight of operator. More abduction than is shown may be desirable.

TABLE 1.—*Anterior Shoulder Dislocations*

	No.
Dislocations (all males)	28
Initial dislocations secondary to trauma	15
Recurrent dislocations associated with marked trauma	8
Recurrent dislocations associated with mild or no trauma	5
No.	
Patients involved	23
Associated fracture of greater tuberosity	2
Age, Yr. — No.	
16	1
56	1
60	1
17–24	20

Duration of time between dislocation and reduction varied from 3 minutes to 2 hours, but more commonly from 25 to 40 minutes.

the affected extremity and places his belt around the forearm just distal to the elbow. With the arm moderately abducted at the shoulder, the body weight of the operator can be applied in line with the humerus until reduction is accomplished. It must be appreciated that considerable traction force can be obtained, and it is believed that this method is not as traumatic as either of the other two methods. Bennett [3] pointed out that this method merely places the arm in the position in which the dislocation originally occurred and reverses the force producing the dislocation.

A brief résumé of the cases encountered is presented in Table 1.

A word about the general handling and the postreduction treatment is appropriate. Prior to the reduction, unless it is on the athletic field, the shoulder is x-rayed to rule out fracture. Two cases in this group had associated fractures of the greater tuberosity. After reduction, if the initial dislocation is associated with trauma, as it usually is, or if it is a second or third dislocation associated with definite, severe trauma, the arm of the affected shoulder is immobilized in a Lund swath for three weeks. The Lund swath is frequently covered with plaster for better security of the dressing. I prefer this dressing to the much more commonly used Velpeau dressing, inasmuch as in a Lund swath the elbow can be left at a right angle and stiffness of the elbow as a post-treatment result is not experienced. After removal of the Lund swath at the end of three weeks, a sling is applied and physical therapy, in the form of heat and active, graduated exercise, is instituted. The sling usually is required only from three to seven days.

Where the dislocation has occurred without trauma, only a simple sling is used after reduction. The patient is then encouraged to agree to a stabilization procedure of that shoulder.

Elbow Dislocations

Since June, 1954, we have encountered 20 posterior dislocations of the elbow. Although the majority have occurred in wrestling, we have experienced these injuries in all sports, as will be evident by examination of Table 2. Anesthesia was not required in a single one of these cases. Lavine [4] has described a simple method of reducing dislocations of the elbow, by resting the arm over the back of a chair. The method I use is believed to be an original one and allows for greater relaxation and more certainty of reduction. It consists merely of placing the patient on a high table in a prone position, with the affected extremity hanging freely over the side of the table. Gentle downward traction is applied at the wrist. All the time necessary is taken, which is usually from 1 to 10 minutes. Here, as in shoulder reductions, forceful traction results in severe spasm and failure. Within one or two minutes of traction the olecranon will be felt to ride distally on the humerus. At this point the operator, while maintaining traction at the wrist with one hand, gently lifts the humerus laterally to flex the elbow about 20 degrees, thus completing reduction (Fig. 4). The reduction in this fashion is so gentle that frequently on lifting the humerus the elbow is found to be reduced

TABLE 2.—*Posterior Elbow Dislocations*

Total number		20
Ages, Yr.		
10		1
18–30		19
Method of injury		
Wrestling	15	
Basketball	1	
Soccer	1	
Tumbling	1	
Football	2	

Fig. 4.—Reduction of elbow with dependent traction. Humerus is lifted laterally with traction maintained on wrist.

and it is not certain when reduction actually occurred.

X-rays are taken prior to reduction to rule out associated fractures. After x-ray verification of reduction, the extremity is immobilized in a posterior splint with the elbow at a right angle and the forearm in neutral position. After three weeks the posterior plaster splint is removed, a simple sling applied, and physical therapy in the form of whirlpool and graduated exercise instituted. Initially, there is considerable stiffness of the elbow. Six weeks to six months, and occasionally longer, may be required to regain full range of motion.

Summary

Acute dislocation of the shoulder and of the elbow in the young or old, in male or female, and in muscular-athletic or less well-developed persons may be reduced without anesthesia and without increased pain or trauma. Methods for accomplishing reduction of such dislocations are described.

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Use of Local Anesthesia in Treatment of Fracture and Dislocations

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The surgeon who is to treat a fracture or dislocation by closed reduction must choose both the proper anesthetic agent and the proper time for reduction. General anesthesia, local anesthesia, or no anesthesia is the choice, and surgeons differ widely in their preference. It is widely accepted that general anesthesia should be given only after hospitalization, routine history taking and physical examination, basic urine and blood studies, and proper premedication, and on an empty stomach. This necessitates availability of a hospital bed and frequently a delay of two or more hours before definite treatment is carried out, and greatly increases the cost of treatment. A study of the use of local anesthesia in fractures and dislocations requiring reduction has been carried out in order to evaluate the proper place of local anesthesia. The obvious advantage of facilitating immediate definitive treatment that can be carried out as hospital outpatient or office therapy makes it desirable to extend the use of local anesthesia as far as possible.

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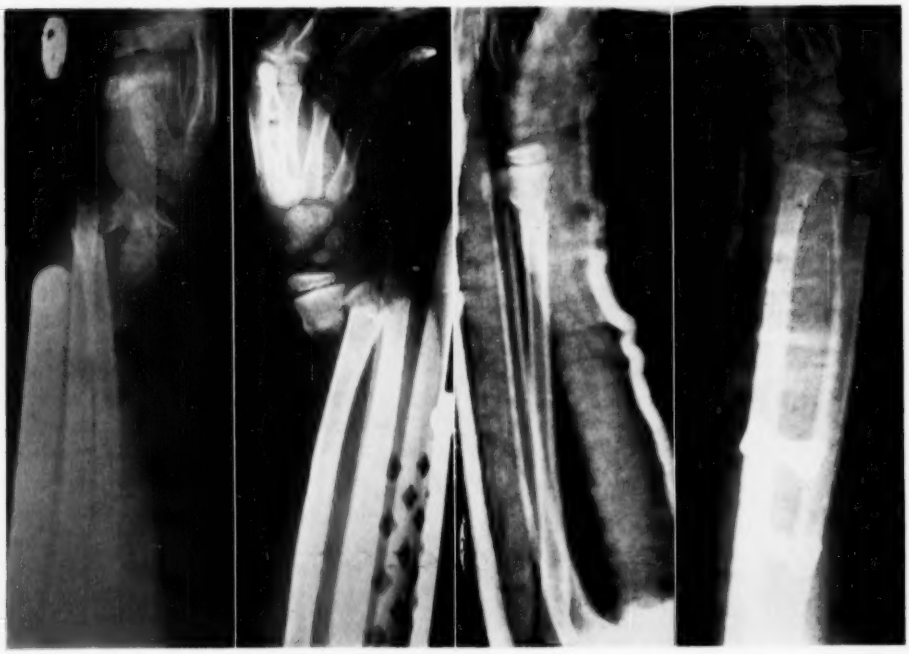
Local anesthesia had its beginning in 1884, when Koller [1] showed that cocaine would anesthetize the cornea. Halsted demonstrated in 1885 that a cocaine solution

Prereduction and postreduction films of fracture in a 6-year-old child treated as outpatient, with local anesthesia. Most such fractures can be so handled.



would temporarily stop conduction when injected around a nerve trunk. Procaine, a relatively safe and satisfactory local anesthetic agent, was synthesized in 1904 by Einhorn. Lidocaine (Xylocaine) was synthesized in 1943 in Sweden by Löfgren and Lundqvist; clinical trial began in 1944 at the Karolinska Hospital. This drug is from a different chemical family than the procaine group, and its pharmacological properties have been described by Goldberg.[2]

Gordh,[3] in 1949, reported the use of lidocaine in 405 general medical and surgical cases. He found the safety coefficient to be four times as high as that of procaine. The drug was completely nonirritating and extremely stable to changes in pH or boiling. Lloyd and Blythe [4] reported on the use of lidocaine in 900 dental administrations. They concluded that block anesthesia was induced faster, was more complete, and was longer-acting with lidocaine than with procaine. It was estimated that 60% as much lidocaine as procaine was used. In 900 administrations they had no major reactions, and the only minor reaction was an episode of syncope. Pitkin [5] states that lidocaine can be used safely in patients known to be sensitive to procaine. Bonica [6] states that, in equal doses, the toxicity of lidocaine is no greater than that of procaine and that its anesthetic index is two to four times that of procaine. Goodman and Gilman [7] state that lidocaine has approximately the same toxicity as procaine in the 0.5% solution but is 50% more toxic than procaine in the 2% solution.

Study Series

This study includes 169 patients with fractures, dislocations, and epiphyseal separations treated by closed reduction and immobilization using lidocaine local infiltration anesthesia. These studies were done during an 18-month period, from September, 1955, through February, 1957. Fractures not requiring reduction were not included. During this period 24 fractures were reduced without any anesthesia; these were primarily in small children, aged 6 years or younger, and were greenstick fractures requiring only a "snap" manipulation. It was felt that general anesthesia was not warranted in these cases, and that injection of local anesthesia would be more upsetting to the child than the quick manipulation.

Injuries Treated with Local Anesthesia

Fractures of one or both bones of forearm in children		60
Colles' fracture in adults		55
Fractures or dislocations of bones of hands or feet		16
Major dislocations		11
Elbow	8	
Shoulder	2	
Ankle	1	
Epiphyseal separations		18
Distal radius	9	
Distal tibia	4	
Proximal humerus	1	
Fractures of shaft of tibia and fibula		6
Supracondylar fracture of humerus		4
Bimalleolar fracture of ankle		2
Fracture of shaft of radius and ulna in adults		1
Total		169

The accompanying Table shows the variety of injuries treated with local anesthesia. In one case reduction was tried unsuccessfully with local anesthesia, followed by successful manipulation with general anesthesia. In three cases reduction was inadequate initially and manipulation was successful a second time without use of additional anesthetic agent. There were five open fractures with small puncture wounds that were treated in the same manner as the closed fractures except for the postmanipulation use of antibiotics. No infections occurred in the series. The age range of the cases is tabulated as follows:

Age, Yr.	No.
4-14	78
15-59	76
60-81	15
	169

Method

The patient was given suitable preliminary sedation or analgesic premedication. If use of a fairly large amount of anesthetic agent was anticipated, a barbiturate was used because of the lessened toxicity of lidocaine in the presence of barbiturate. The intended procedure was carefully explained to all patients, including children. The parents of children were invited to be present if they desired, and their presence usually made it easier for the child to accept local anesthesia.

The skin of the injured area was prepared by gentle scrubbing with an antiseptic soap

solution. A skin wheal was made with a hypodermic needle, using either 1% or 2% lidocaine hydrochloride without epinephrine. The wheal is used for observation of any type of immediate unfavorable reaction to the drug, as well as an anesthetized area for introduction of a larger needle into the fracture or dislocation hematoma. Effective anesthesia is dependent upon injection of the agent into the hematoma; this is usually not difficult and does not require a large volume of the agent. Lidocaine hydrochloride 1% was used in the children and 2% in the adult. In areas of relatively slight soft-tissue mass, such as the wrist, an attempt was made to aspirate the same volume of blood from the hematoma as was used of the anesthetic agent. This was done by drawing blood back into the syringe and injecting the mixture of blood and lidocaine back into the hematoma several times without moving the needle. With this technique the fluid mass at the fracture site is not increased. Epinephrine was not used in the lidocaine because it offers no advantages and may produce an unfavorable reaction. Remanipulation, when required, was done quite satisfactorily without additional injection and was carried out as long as 40 minutes after the original injection. Not more than 10 cc. of 2% lidocaine hydrochloride was used in any case, and in most cases considerably less. A waiting period of three or four minutes usually is sufficient to permit full anesthesia to develop. Inadequate anesthesia is almost always due either to faulty technique or to clotting of the hematoma.

This anesthesia technique is dependent upon diffusion of the agent through the liquid hematoma. If the hematoma is small or if the blood has clotted, it may be difficult to get satisfactory anesthesia without wide infiltration. In general, the earlier a fracture is treated after injury, the easier it is to get effective anesthesia. The majority of cases in this study were treated definitively from one to six hours after injury.

In this series only two major reactions were encountered. Both these were a temporary partial vascular collapse. These reactions lasted not longer than 10 minutes and no specific treatment was necessary. It is advisable to be prepared to treat serious reactions. One should have available oxygen and an intravenous vasoconstrictor to handle a vascular collapse, an intravenous barbiturate to manage convulsions, and an intravenous antihistaminic to treat severe urticaria.

Summary and Conclusions

A series of 169 patients with fractures, dislocations, and epiphyseal separations treated by closed reduction using local anesthesia is described.

The principles and technique of this method are discussed.

Lidocaine hydrochloride solution, 1% or 2%, without epinephrine is considered to be the anesthetic agent of choice in this field.

Excellent anesthesia is the rule when this method is properly carried out.

Local anesthesia in the majority of fractures and dislocations requiring closed reduction is safe, effective, and economical of time and facility.

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Intramedullary "Pull-Out" Wire Fixation in Surgery of the Hand

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One of the major problems in reconstructive surgery of the hand, whether it be traumatic or elective, is adequate fixation of the injured part. This fixation should be nonspace-taking and nonirritating to already traumatized tissues. Another criterion to be met is the institution of early mobility of uninjured segments of the hand and the reinstitution of mobility of the injured part as soon as skeletal structures are stabilized and soft tissues have healed. The third criterion to be observed is the prevention of postoperative hematoma and edema. This last problem I have met only by the use of an adequate, carefully applied pressure dressing. A cast permits, and even encourages, capillary oozing and is difficult to apply uniformly while attempting to maintain the position of injured parts.

Kirschner-wire fixation of phalanges, metacarpals, and carpals has proved an excellent solution to this problem, and one which has fulfilled the criteria noted above.

Steinmann pins and Kirschner wires have been used extensively, since their introduction in 1909, for the fixation of fractures and for stabilization of arthrodesed joints. However, there has been understandable reluctance to place intramedullary pins across joints, and this reluctance has extended to the use of intramedullary wires in hand surgery. Watson-Jones [9] emphatically cautions against the transarticular placement of pins or the use of metal adjacent to joints and states that limitation of motion of the joint is an invariable sequel. However, in my experience, no specific loss of function has been noted with their use in the digits, and in no instance has ankylosis resulted from the transarticular use of the wire per se. Employment of internal pin fixation has been reported by Jordan,[3] who considered the use of pull-out pins primarily in long-bone fractures, and by Moore,[4] who employs pull-out pins, by placing them transversely to the axis of long bones in treatment of shaft fractures. Murray,[5] Waugh and Ferrazzano,[7] and vom Saal [8] have described the use of Kirschner wires in treatment of specific fractures about the hand. Bunnell,[2] in his text, has noted in photographic captions that Kirschner wires may be employed to maintain unstable fracture-dislocations of the hand and in arthrodesis of phalangeal joints. Pratt [6] has advocated the use of Kirschner wires in the treatment of avulsion injuries of the extensor tendons at the distal phalanx. However, the predominant number of reports in the literature have concerned the use of Kirschner wires transosteally in providing skeletal traction or rigid immobilization of fractures. The technique of closed fixation of metacarpal fractures, as advocated by Berkman and Miles [1] and others, can be exceedingly difficult and requires close x-ray supervision and excellent assistance. In my experience, reduction of fractures of the bones of the hand and maintenance of reduction by skeletal traction is time-consuming for the surgeon and economically wasteful for the patient, and requires extremely close supervision, with uncertain end-results. I employ pull-out wire fixation for all displaced fractures of the phalanges, metacarpals, or carpals, in the stabilization of arthrodesed joints of the hand, and where splinting of the digits is

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desired after release of contractures. The incidence of the postoperative complications of edema and hematoma has been materially reduced by the use of this method. When it has been used in compound fractures, no postoperative infections have developed secondary to the use of internal fixation and the complications of skin necrosis from attempts to immobilize fractures in plaster have been avoided.

Injuries of the Carpus

Fractures of the bones of the carpus, other than the navicular (scaphoid), are relatively uncommon, and yet, when they occur, are usually the result of direct trauma with subsequent displacement of fragments. Anatomical reposition is mandatory if wrist function is to remain unimpaired and painless. Manipulative reduction is usually unsatisfactory, and, for this reason, open reduction and fixation with Kirschner wires has been employed. Fusion of the wrist joint in the treatment of traumatic arthritis can best and most simply be achieved by the use of inlay iliac bone inserted into a trough cut into the radius and secured to the distal carpal row, accompanied by exeresis of cartilage from the radial one-half of the carpus. Fixation of the proximal aspect of the graft into the radius usually proves relatively simple. However, maintenance of contact of the distal end of the graft frequently is troublesome and can readily be solved by use of an obliquely placed transfixion Kirschner wire. In wrist fusions performed by this method, external fixation has not extended beyond eight weeks, and in all instances solid bony union has resulted.

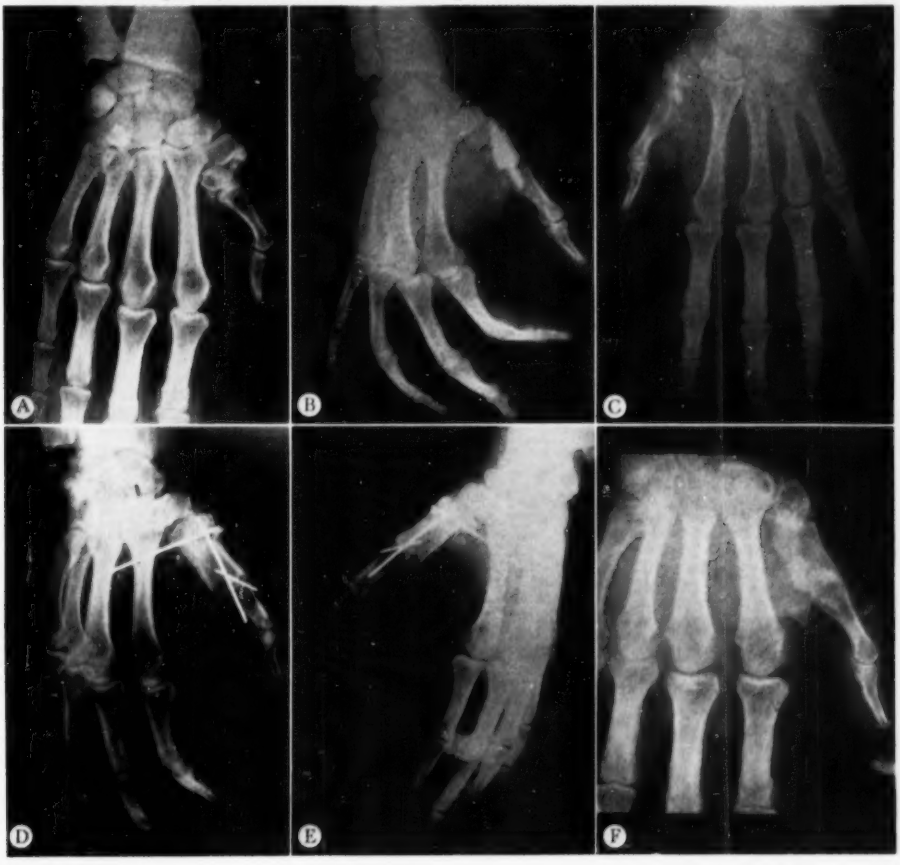
Injuries of the Metacarpals

In the treatment of metacarpal injuries, the Kirschner wire employed is inserted retrograde through the head of the metacarpal and then drilled across the defect, under direct vision, down the proximal medullary canal and permitted to resurface just proximal to the carpus. The pin is drilled retrograde with the metacarpophalangeal joint in full flexion and is countersunk beneath the articular surface. Motion of the involved digit is instituted in 10 days from a light volar splint, and full motion is permitted at 3 weeks. Use of digital traction for a period adequate to permit stability of fractured metacarpals usually requires six to eight weeks and is time-consuming for both patient and physician, since the traction device must be carefully observed to prevent distraction and delayed union. It is usually impossible for the patient to work, and dressing and ordinary daily activities are limited by the use of the traction appliance. Mobilization of the involved digit after the use of traction usually requires at least as long as the period of immobilization. Where gross comminution of metacarpals exists, intramedullary fixation to maintain alignment has been found effective when combined with transverse fixation of the fragments to the adjacent metacarpal. Use of the transverse pins alone, as advocated by Berkman and Miles,[1] frequently results in angulation of the fragments, and this difficulty has been overcome by the use of the longitudinally placed pin. Use of Kirschner wires in Bennett's fracture has been reported by Wiggins and Bundens,[10] and is felt to be an excellent method of achieving stabilization of a difficult fracture-dislocation if manipulative attempts are unsuccessful or if difficulty is noted in maintenance of postoperative traction or immobilization. I have performed metacarpophalangeal arthrodesis of the thumb with increasing frequency for a number of pathologic conditions. 1. In opponens paralysis, where there is a combined extrinsic muscle imbalance, metacarpophalangeal arthrodesis is performed as an initial stage, followed by tendon transplants. 2. Patients with rheumatoid arthritis, with otherwise minimal involvement of the hand, frequently present subluxation of the metacarpophalangeal joint of the thumb, with weakness of pinch and pain on grasp. In these patients, metacarpophalangeal arthrodesis has been found

to be an effective method of restoring stable pinch, grasp, and opposition without pain. 3. Metacarpophalangeal arthrodesis of the thumb is employed in providing "automatic" hands for quadriplegics, where the tenodesis effect of the extrinsic muscles is utilized to provide grasp and pinch. Stabilization of the thumb is a requisite in these cases, and early mobilization of the hand is desired to prevent loss of tone of wrist extensors. Routinely, in arthrodesis of this joint intramedullary Kirschner wires have been employed for fixation. The hand is immobilized postoperatively in a pressure dressing, which is removed after five days and replaced with a well-fitting cast. The cast is maintained for an additional 1½ weeks, at which time the patient is permitted to begin movement from a bivalved cast which is maintained for an additional 10 days. In all instances, clinical union of the arthrodesis has been noted at six weeks and full active use of the hand has been permitted at that time. Use of intramedullary pins, rather than obliquely placed pins, is advocated, since it permits compression of bone ends and prevents distraction resulting from absorption of bone.

Following collateral ligament resection and volar capsulotomy for extension contractures of the metacarpophalangeal joints, the metacarpophalangeal joint is immobilized in

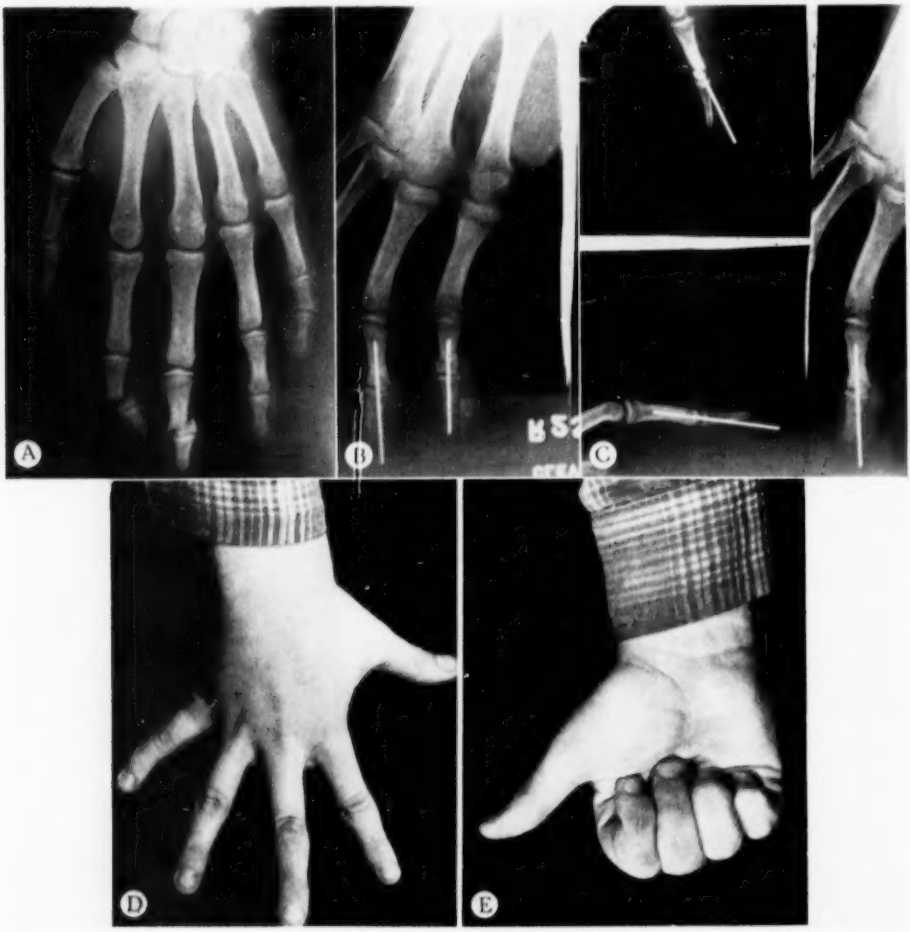
Fig. 1.—*A*, remote fracture of metacarpal with gross absorption of fracture fragments. *B*, reconstruction employing key graft, followed by plaster immobilization. *C*, non union and resorption graft. *D* and *E*, free iliac graft with multiple pull-out pins. Note pin maintaining thumb-web length. *F*, end-result 16 weeks after graft.



60-degree flexion by the use of pull-out, transarticular wires, which are removed at the end of two weeks to permit metacarpophalangeal motion. Splinting of the hand, with the danger of pressure necrosis, is thus avoided, and early motion of the proximal interphalangeal and distal interphalangeal joints is possible.

Web-space contracture is a common and extremely troublesome complication following severe injuries to the hand. Prophylactically, I have employed transmetacarpal wires, placed between the first and second metacarpals, to maintain maximum thumb-web width and to prevent the development of contracture. Following release of thumb-web contractures and either full-thickness skin grafting or pedicle grafting to the thumb web, transmetacarpal wire fixation has been employed, with excellent maintenance of position of the released thumb in a situation where no adequate alternative method exists. In reconstruction of metacarpals, Kirschner wires have been employed as a "scaffold" and have provided firm fixation of the free graft (Fig. 1). In pollicization of digits, intramedullary wire fixation is mandatory if skin necrosis is to

Fig. 2.—*A*, power-mower injury, resulting in compound fractures with gross soft-tissue injury. *B* and *C*, post operative reduction and fixation with pull-out wires. *D* and *E*, function one year later.



be avoided and adequate pressure permitted on the skin flaps. In these cases any shift of position of the transplant may be sufficient to permit arterial damage and loss of the transplanted digit.

Injuries of the Phalanges

In treatment of injuries of the phalanges where there is extensive soft-tissue damage, or where instability of fracture fragments exists, 0.015 Kirschner wires have proved effective in permitting early skin healing and excellent maintenance of fracture fixation. As in the case of the metacarpals, the wire is placed retrograde and then inserted up the medullary canal of the proximal fragment to the adjacent joint (Fig. 2). The wires are permitted to protrude and are not covered except by a strip of tape to prevent their catching the patient's clothing or other articles (Fig. 3).

Kirschner-wire fixation has been combined with open repair of all avulsion injuries of the extensor tendon at the distal phalanx. I have not attempted to maintain the proximal interphalangeal joint in flexion after open repair. The distal interphalangeal joint is fixed in maximum hyperextension,

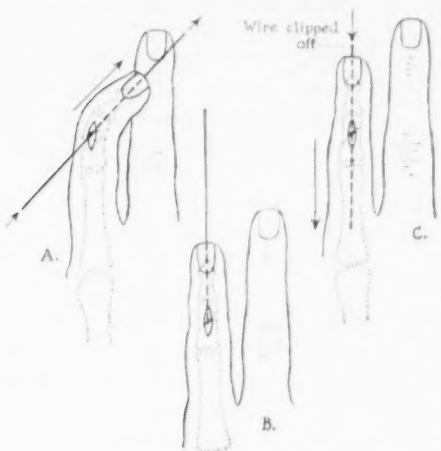


Fig. 3.—*A*, wire inserted at fracture site and across the distal interphalangeal joint. *B*, fracture, reduced and wire passed retrograde to proximal fragment. *C*, wire firmly secured in proximal fragment and passed across proximal interphalangeal joint.

and in the tendons thus repaired full function has been restored. Again, in this instance, the wire is permitted to protrude, and it is advised that the wire be left at least ⅛ in. exterior to the pulp of the finger to facilitate easy removal.

In those cases where the condition of the skin of the hand precludes early splinting, following release of intrinsic plus contractures of the proximal interphalangeal joints, 0.015 Kirschner wires have been employed transarticularly for 10 days after surgery to permit early mobilization of the metacarpophalangeal and distal interphalangeal joints. The wires are inserted across the proximal interphalangeal joint, with this joint maintained in 45-degree flexion, and with the distal interphalangeal joint in maximum flexion, thus permitting full flexion and extension of the distal interphalangeal joint, even though the pin remains in situ. In rheumatoid arthritis, where broad grasp is desired, and destruction of joint surfaces has occurred at the proximal interphalangeal joint with subluxation, I have performed interphalangeal arthrodesis and maintained fixation of the joint by the use of Kirshchner wires. The technique is the same as that employed in the proximal interphalangeal joint following intrinsic plus release and has permitted early mobilization of the distal and metacarpophalangeal joints.

Summary

Use of Kirschner wires is advocated where stabilization of bone surfaces is required and where maintenance of joint position is necessary following release of fixed contractures. The advantages of Kirschner wire fixation are as follows: 1. Early mobilization of the uninjured parts with maintenance of function and without loss of position. 2. Application of an adequate pressure dressing is possible, thus preventing hematoma formation and edema. 3. Following extensive trauma, maximum stability of the injured parts promotes soft-tissue healing and permits early mobilization of the hand. 4. Transarticular placement of the pin

has not resulted in ankylosis or limitation of motion when cartilage surfaces of the joint were intact. 5. Postoperative care of the hand is minimized for both patient and physician and permits earlier return to full function without loss of the desired end-result.

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Treatment of Fresh Fractures of Neck of Femur with Intramedullary Stem Prostheses

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Fractures of the neck of the femur in aged people in poor general condition have in many cases continued to offer a poor prognosis. It is not the purpose of this paper to present the various mechanical factors that serve to fracture a hip, nor is it to call to attention the methods of diagnosis of a fractured neck of the femur. All this is very well known. Our primary purpose is to report our experiences in 45 selected cases of fresh fracture of the neck of the femur treated by the insertion of an intramedullary metallic stem prosthesis. In most of our cases the patient was considered too old or too feeble or too fragile and the fracture to be such that in our opinion the interest of the patient would best be served by not treating him by the conventional hip-nailing. A few of the patients were treated by this method because of our failure to accomplish a satisfactory closed reduction before an intended hip-nailing. We share the opinion of many other orthopedists in that fracture of the neck of the femur in a feeble or fragile patient very commonly serves as a terminal event. We further are of the opinion that the higher the fracture in the neck, the poorer becomes the prognosis for healing. The insertion of the metallic prosthesis in fresh fractures of the neck was undertaken with the expectation of reducing the incidence of such complications as uremia, hypostatic pneumonia, shock, and death. In addition, it was hoped that the operation would decrease the amount of postoperative pain, the length of hospital stay, and the period of non-weight-bearing. Feeble and fragile patients generally pose a nursing problem; consequently, it was hoped also that the operation would make the nursing care easier for both the patient and the nurse.

We wish to be extremely emphatic in conceding that a successful hip-nailing is more desirable and gives a much better end-result than any replacement prosthesis. However, we also have felt that many femoral-neck fractures in feeble and fragile elderly people very often lead to unsatisfactory end-results. Early ambulation is the prime objective of operative intervention in these cases.

Requisites for Replacement Prosthesis

We feel that there are certain requisites which must be met before undertaking immediate replacement prosthesis. The requisites which have been the major ones in our series include the following:

1. The surgeon must feel reasonably sure that the patient will be able to walk again after the operation.

2. The fracture should preferably be of a high femoral type, and the patient should be feeble and aged.

Midneck fractures of persons in poor general condition which cannot be satisfactorily reduced should be so treated.

Advantages

Some of the advantages of doing replacement prostheses as a primary operation are as follows: (1) early ambulation; (2) reduction in the number of secondary reconstruction operations; (3) facilitation of nursing care to both the patient and the nurses; (4) reduction of length of hospital or convalescent-home stay, thereby reducing the cost of treatment.

Fig. 1.—AP view, showing high femoral-neck fracture.

We have undertaken the use of the replacement prosthesis in selected cases, notwithstanding the opinion of some orthopedists that the commonest abuse in the use of endoprosthesis is in fresh fractures of the femoral neck. We have not allowed our judgment to be influenced by the immediate functional or economic advantages of the operation. We have also been aware that it is the feeling of some orthopedists that an endoprosthesis is justifiable only as a salvage procedure, when a high femoral-neck fracture fails to heal or undergoes aseptic nercosis after apparent healing. Past sad experiences dictated to us the advisability of doing our replacement prosthesis operations as a primary procedure in fresh fractures in cases which met certain qualifications.

We have reviewed 45 selected cases of fresh femoral-neck fractures admitted to the Rhode Island Hospital since January, 1954, in which the type of fracture and the condition of the patient were such that success by routine hip-nailing appeared dubious (Figs. 1-3). Over the same period of time, 183 patients with femoral-neck fractures were admitted to the fracture service of this hopsital. This number does not include the intertrochanteric or subtrochanteric fractures. Endoprosthesis operations were done on approximately 25% of all patients with neck fractures admitted.

Of the 45 patients, 44 were women and 1 was a man. The youngest woman was 51 and the oldest was 92. The 51-year-old patient was operated upon because of failure to reduce satisfactorily her displaced sub-

Fig. 2.—Lateral view, showing high femoral-neck fracture.

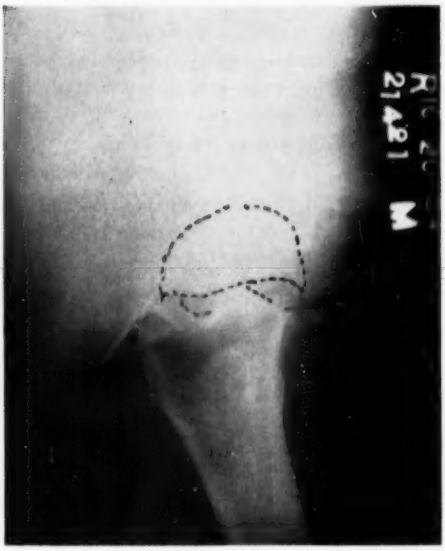

Fig. 3.—Fractured head and most of neck have been removed and have been replaced by the Austin Moore intramedullary stem prosthesis.

capital fracture and because of her poor general condition. The over-all age of the patients was 78.38 years. There were 28 sustained fractures of the left hip and 17 fractures of the right hip. We are unable to give a satisfactory explanation as to why the ratio of fractures of the left hip to fractures of the right hip was almost 2:1, except that all of our patients were right-handed; consequently, the left side of the body would be expected to be weaker than the right side.

With reference to the site of the fracture, 33 of our cases represented subcapital fractures without impaction. The remaining 12 fractures were of the midneck type. The operation was done anywhere from 2 to 18 days following the injury, with an average of 5.84 days.

We used the Austin Moore self-locking type of Vitallium intramedullary stem prosthesis in all our cases. We are of the opinion that this type of prosthesis preserves neck length, is less apt to become angulated than many other types, is less likely to become loose, with bone absorption, and is less apt to break.

Approach

The operation in all our cases was done by the posterolateral approach as used by Moore.[19] We felt that this approach was desirable because of the simplicity of reaching the joint surgically and because of the simple immediate postoperative care required.

With the patient under spinal or general anesthesia and lying on his side with the fractured hip up, proper preparation and draping is done. The injured leg is encased in sterile sheets or a long sterile stockinette so as to make for easy manipulation of the leg as needed. The incision is started about 5 in. (12.5 cm.) below the posterior superior spine and then carried downward and outward to 3 or 5 in. (7.5 to 12.5 cm.) below the greater trochanter. All bleeding points are clamped and tied. The gluteus maximus muscle is split by blunt dissection along the course of its fibers. Either self-retaining or manual retractors are applied. The sciatic nerve is easily exposed by blunt dissection. A rubber dam is passed around it and it is retracted. If difficulty is encountered in locating the sciatic nerve, a search for the piriformis muscle is made, and the sciatic nerve will be found to be coming out from beneath the piriformis muscle. Occasionally it becomes necessary to divide the abductor group from their attachment to the greater trochanter. The gemellus superior, obturator internus, and gemellus inferior are identified and severed about ½ in. (1.3 cm.) from their insertion into the medial surface of the greater trochanter. These muscles are then peeled backward, exposing the capsule of the hip joint. The capsule is then divided longitudinally at its femoral margin, exposing the hip joint. The hip joint is then dislocated by flexing, adducting, and internally rotating the extremity. In most cases the head of the femur can be lifted out of the acetabulum with a large towel clip or with a Steinmann pin. Any remaining portion of the neck is removed down to the base of the neck. The remaining neck and the shaft of the femur are hollowed out by means of a special rasp. The heads of two or three prostheses, of varying sizes, are tried in the acetabulum to determine the best-fitting head. The fenestrations in the stem of the prosthesis are packed with bone chips, and the prosthesis is then driven into place so that its fin will be toward the dorsal surface of the greater trochanter. By taking this particular step, anteversion of the neck can be preserved. At times it becomes necessary to make a small longitudinal slot on the dorsal surface of the greater trochanter in order to get a better fitting of the prosthesis in the greater trochanter. The acetabulum is washed out with hot saline, and the hip is then reduced and motion-tested. In order for a good result to come about, the patient should have a full range of joint motion at this time. The capsule is now closed with interrupted chromic 0 absorbable surgical sutures, and the gemelli and obturator internus are resutured to their origin with interrupted chromic 0 sutures. The rubber dam is removed from the sciatic nerve; the gluteus maximus muscle is approximated by means of loose interrupted chromic 0 sutures; the subcutaneous tissue is closed by continuous plain 00 absorbable surgical (gut) sutures, and the skin is closed with black silk.

Results

Immediate Postoperative Immobilization.—Thirty-nine patients were treated by means of Buck's extension with a weight varying from 3 to 5 lb. The remaining six patients had their leg partially immobilized by sandbags placed on each side of the leg.

Weight-Bearing.—This was allowed anywhere from 1 to 42 days following operation, for an average of 13.1 days. In the

earlier cases the average patient was kept in bed approximately three weeks postoperatively; however, with the gaining of experience with this problem, patients have been allowed out of bed much sooner than three weeks. The hospital stay ranged from 18 to 82 days, in one case, for an average of 37.71 days. The patient who remained in the hospital 82 days had difficulty in balancing herself for several weeks following the operation.

Postoperative Pain.—At the time of the interrogation 34 patients stated that they had no pain whatever on weight-bearing. Nine stated that they had mild pain, and two stated that they had moderate pain. In the two who had moderate pain, we could not be sure of the cause of the pain. Of the 11 patients who reported pain, all had the pain in the hip, and 1 also had pain in the knee on the affected side. Of the 11 who had pain on weight-bearing, 8 stated that they had no pain whatever on non-weight-bearing, while 3 had constant pain.

Range of Motion.—Nineteen patients had a complete range of motion. In these cases the motion in the affected hip was the same as in the unaffected hip. These results we classify as excellent. Nine of the patients had limitation of internal rotation only. Nine had limitation of both internal and external rotation. Eight of the patients in our series had a slight amount of limitation of abduction, adduction, and internal and external rotation. To recapitulate, then, insofar as motion is concerned, the results in 19 cases were excellent, in 18 good, and in 8 fair.

Walking Aids.—Thirty-eight of our patients stated that they used a cane on walking, while seven stated that they used no form of walking aid. Most of the 38 who used a cane stated that they could possibly get along without the aid of a cane but they felt surer of themselves if they used a cane. Of the 38 who used a cane, 2 stated that they used crutches when they went on long walks, as with crutches they did not tire as easily.

Length of Extremities.—Thirty-eight of the patients reported showed no discrepancy in leg lengths. Five had from a ½ to ¾ in. decrease in the length of the hip operated on, while two had a ½ in. increase in the length of the leg treated surgically. It is felt that in the cases where shortening took place, perhaps too much of the neck was removed at operation, whereas in the two cases where there was increased length it is thought that not enough of the neck was removed at the time of surgical intervention. We have learned that if correct leg length is to be obtained, reduction should be accomplished with only slight difficulty. If the reduction is difficult, the neck has been left too long. If the reduction is extremely easy, too much of the neck has been removed.

Discharge Rate.—Eighteen of our patients were discharged to nursing homes. In most of these cases the patient could not go home because there was no one there who could help look after them. The remaining 27 cases were discharged directly to their own homes from the hospital.

In each case the patient was asked if he or she was satisfied with the result obtained, and the patient always answered in the affirmative.

Complications.—One patient developed a foot drop, which cleared up at the end of the third week. Perhaps the foot drop may have been due to undue tension when the sciatic nerve was retracted. Another of our patients developed an acute gallbladder attack, and a gangrenous gallbladder was removed 10 days after the hip operation. Three patients developed decubitus ulcers over the sacral region, which cleared up after the patients had been up and about several days. One patient complained of localized low back pain following the operation. This patient had had spinal anesthesia but also had considerable osteophytosis of the lower spine. The pain cleared up after several days. One patient had difficulty in balancing herself when allowed to be up and about. This cleared up after several

weeks, and she was finally able to get about with the aid of a cane.

Dislocations.—We had no dislocations. We were very pleased with this observation, as we had had dislocations in previous cases, in which a Judet nylon prosthesis had been inserted by the Smith-Peterson approach in cases other than fresh fractures. We have also had dislocations in cases done for ununited fractures of the neck of the femur.

Deaths.—We had no operating-room deaths, and we had no deaths before hospital discharge; however, one patient died four months after the operation of bronchopneumonia. Three died of arteriosclerotic heart disease—one 5 months, one 6 months, and one 14 months after the operation. Another patient died 11 months after the operation of bronchopneumonia. This represents a total of five deaths, the postoperation time ranging from 4 to 14 months. In each case the patient had been discharged from the hospital and was already walking with a cane at the time she was taken with the illness that resulted in her death.

Follow-Up Period.—The shortest follow-up was in the case of one of our patients who died of bronchopneumonia four months after the operation. The longest has been a 36-month follow-up period. The average follow-up length is 17.76 months.

Summary and Conclusions

It is emphasized that in routine hip fractures a successful hip-nailing is more desirable and gives a better end-result than a replacement prosthesis. However, we feel that in certain selected cases of neck fractures, the nailing of the hip by the conventional method carries a calculated risk with it insofar as undesirable results are concerned. We report 45 cases of selected fresh femoral-neck fractures treated by the insertion of an endoprosthesis. The advantages of the operation as a primary procedure include early ambulation, reduction of hospital stay, reduction in the cost of the injury, reduction of complications, and easier nursing care. All patients were operated upon by the posterior approach, with no major complications. Most of our patients have been able to take care of themselves in their homes, using a cane as an aid.

We wish emphatically to restate that, whenever possible, fractures of the neck of the femur should be treated by routine nailing. However, we also feel that the insertion of an endoprosthesis in selected cases of fresh fracture of the neck of the femur has a definite but limited place in the treatment of this type of fracture.

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Treatment of Fractures of the Os Calcis

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No panacea exists for fractures of the os calcis. Many methods of treatment have been proposed because of deficiency in the end-results and because of confusion over classification. Classification of these fractures is simplified by regarding the state of the subastragalar joint as the essence of the pathologic condition. I believe that open reduction, after the method of Palmer, is the treatment of choice for most fractures involving severe displacement in the posterior subastragalar joint or of it.

There are numerous methods of treating "smash" fractures of the os calcis. They vary from early mobilization [2-4] to excision.[13] A summary of the world literature prepared by Widén [18] contains 155 references. One reason for this large number of modalities is the end-result: A treatment which works well is soon adopted. A second reason is that older classifications which have been proposed are frequently involved and are not based on significant elements. Recent methods are simpler and based on more pertinent factors.[5,14,15] A third reason is the comminuted appearance of the os calcis in roentgenograms of fractures which involve the posterior subastragalar joint ("smash" fractures). This appearance makes the surgeon hesitate to apply the principle of restitution of joint surfaces, although the comminution does not actually involve the joint per se.

Classifications of fractures of the os calcis are numerous. However, for practical purposes, these injuries can be divided into those involving and those not involving the posterior subastragalar joint. Injuries in each of these categories can be further subdivided into those which need operative treatment and those which do not.

Any classification of fractures of the os calcis requires as its basis perfect roentgenograms. Cases of fracture of the os calcis should be examined radiographically with an anteroposterior view of the ankle to demonstrate, if present, piling up of bone of the os calcis under the fibular malleolus; a lateral view of the ankle and foot; an anteroposterior view of the foot to demonstrate possible involvement of the calcaneocuboid joint, and an axial view of the heel. At times the opposite side may have to be x-rayed for comparison.

Fig. 1.—The central ray must pass tangentially through the subastragalar joint.

The axial view presents the greatest technical difficulty. In this view the central ray must pass tangentially through the subastragalar joint (Fig. 1). This can be done either with the patient standing and the central ray passing from the back of the heel toward the sole or with the patient recumbent and the central ray passing from the sole to the back of the heel. A technically satisfactory axial view of the heel shows on the lateral side of the foot the base of the fifth metatarsal and the posterior subastragalar joint, and on the medial side of the foot the joint between talus and sustentaculum tali (Fig. 2).

The immediate care of the patient with a fracture of the os calcis is important. First, one should rule out injuries of other

Fig. 2.—To be noted are the base of the fifth metatarsal, the posterior subastragalar joint, and the joint between talus and sustentaculum tali.

parts of the body, particularly a compression fracture of the spine. Accordingly, the patient should be examined and roentgenograms made of suspected regions. Severe fractures of the os calcis are usually accompanied by tremendous swelling of the foot and ankle. This can be well controlled by applying copious amounts of sheet wadding, followed by a loosely applied elastic bandage and considerable elevation. Applying ice is

Fig. 3.—Skin necrosis threatened; open reduction performed.

helpful, but heat is contraindicated in the first 24 hours. After five to seven days, the swelling has usually subsided sufficiently to permit carrying out specific treatment.

One's specific treatment should be dictated in the main by the roentgenographic findings. Mild fragmentation of the tuber of the os calcis without joint involvement, the result of a direct blow, can be treated by brief plaster immobilization. On the other hand, an injury with considerable displacement of a portion of the tuber, which exerts pressure on the skin and threatens to cause necrosis, requires manipulation or open operation (Fig. 3). The usual routine with patients who do not require operative intervention is to immobilize the foot and ankle in a short leg cast for three to six

Fig. 4.—Lateral (*A*) and axial (*B*) roetgenograms of fracture involving posterior subastragalar joint with minimal displacement.

weeks, followed by non-weight-bearing, mobilizing activities, and then free weight-bearing in a shoe which is appropriately padded.

Fractures which involve the posterior subastragalar joint with minimal displacement are also treated in that manner (Fig. 4*A* and *B*). Fractures with severe involvement of the posterior subastragalar joint are notorious for the prolonged temporary and severe permanent disability which they cause the patient. Three common methods for treating this fracture are early mobilization,[2-4] the method of Boehler (or a variation thereof),[9] and early arthrodesis.[7,8]

It is difficult to understand the rationale behind the method of Boehler or the method of early mobilization. There are no fibrous structures passing between the articular surfaces of the posterior subastralagar joint which can effect traction and hence reposition of the depressed portion of the os calcis in the Boehler method. No attempt is made to restore the joint surface in the method of early mobilization.

However, it has been noted many times that with these methods, even without triple arthrodesis, there eventually results a satisfactory foot.[1,5,6] This occurs when a firm fibrous union eventuates. However, it takes about 18 months for the union to occur. Until it does, the affected patient has painful feet and great disability. The foot frequently remains deformed, with no arch and a broadened heel. Further evidence that fibrous fusion with alleviation of pain occurs spontaneously is contained in my experience, as well as by the fact that few patients who have had old fractures of the os calcis are seen by orthopedic surgeons for therapy years after their initial injury.

Arthrodesis leaves a broadened heel and a flat foot.

Open reduction of certain fractures of the os calcis can shorten the duration of the disability and often results in a foot approaching normal in both function and appearance.[5,12,14,15,18]

Whittaker called the attention of the American surgeon to open reduction of the os calcis.[16] Palmer, in 1948, brought it to the further attention of English-speaking orthopedic surgeons.[12] Open reduction had been done as early as 1921 by Leriche.[11] Lenormant added a bone graft to support the articular facet.[10] Instrumental methods to replace the depressed portions of the posterior subastragalar joint had been used by Westhues in 1934.[16] He passed a nail through the tuber to elevate the depressed portion of the joint. Some surgeons have hesitated[9,19] to attempt open reduction of the os calcis, principally, I feel, because of the appearance of great comminution in roentgenograms of these fractures. The comminution involves the body, and not the articular surface: Open reduction is feasible and desirable.

When a fracturing blow strikes the heel, the fracture occurs in the os calcis between the tuber and the sustentaculum. This fracture line frequently extends into the posterior subastragalar joint. If the force continues with the weight from above, as in a fall, or with pressure from below, as when the heel is broken from a rising surface (for example, an explosion), the lateral fragment of the os calcis continues to advance against the talus, and subchondral compression of the os calcis results. With recoil, a ledge is left in the posterior subastragalar joint[5,12,14] (Fig. 5). To correct this ledge, the compression is relieved by open reduction, elevation and position being

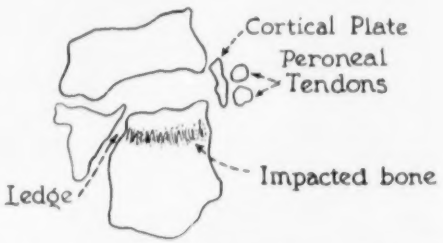


Fig. 5—The ledge may be on the facet of the os calcis in the posterior subastragalar joint, may be a "channel" if the compression is central, or may be medial to the joint surface. (Modified after Palmer.[12])

maintained with a bone graft. When the entire facet is depressed, it, too, is replaced by open reduction.

Modifications of Palmer's technique have been found desirable. The patient is placed on a regular operating table with the involved side elevated or in a prone position. A Kirschner wire is placed through the tuber of the os calcis and held in a sterile bow. The incision is of a short Kocher type. Further dissection carries one to the peroneal tendons, which are reflected with their sheaths anteriorly. Due respect must be paid to the sural nerve, which runs almost in a line of the incision and which is visualized and carefully retracted. It is necessary to cut the calcaneofibular and anterior talofibular ligaments to obtain ingress to the posterior subastragalar joint. This joint is inspected. The usual finding is that the facet of the posterior subastragalar joint is in two pieces and that the lateral piece is depressed. At times the entire facet is depressed, or there is a central depressed portion.

An elevator is placed through the aperture made by the buckling of the lateral wall of the os calcis under the depressed portion while traction is being maintained on the heel via the Kirschner wire. The depressed portion of the posterior facet of the os calcis is elevated, and position is maintained by a bone graft placed in the resulting defect. "Homogeneous" rather than autogenous bone is used to avoid further disability in a donor site. This practice is felt to be very important in industrial cases.

The joint is then inspected and, if possible, the ledge or ledges visualized. This visualization is difficult. A pitfall is that by packing the bone into the defect a diastasis of the joint surface may occur. Hence roentgenograms are made in the operating room to assure that a good reduction has been obtained. If it has not, any displacement is corrected (Figs. 6A-B, 6C-D, 6E-F). If the articular surface itself is comminuted, as it sometimes is, elevation of the fragments permits earlier fibrous fusion and shorter disability. The tourniquet which is

Fig. 6A-B.—Axial and lateral views of "smash" fracture of the os calcis.

Fig. 6C-D.—Roentgenograms made in operating room. Note the incomplete reduction and the diastasis of the joint surface.

always used is released and hemostasis secured. The wound is then closed in layers.

A long leg plaster cast, with the knee at 45 degrees to relax the gastrocnemius muscle, is applied with a rolled piece of felt molded under the fibular malleolus to prevent spread of the lateral portion of the fractured os calcis. The pin is incorporated in the cast. Even when good reduction is not obtained because of comminution of the articular facet, which does occur, the end-results are most satisfactory. The severe

Fig. 6E-F.—Improvement in reduction as seen in roentgenograms made six months postoperatively.

Fig. 7A-B.—*A*, the average result, showing minimal broadening of the heel on the right (operative) side. *B*, the arches.

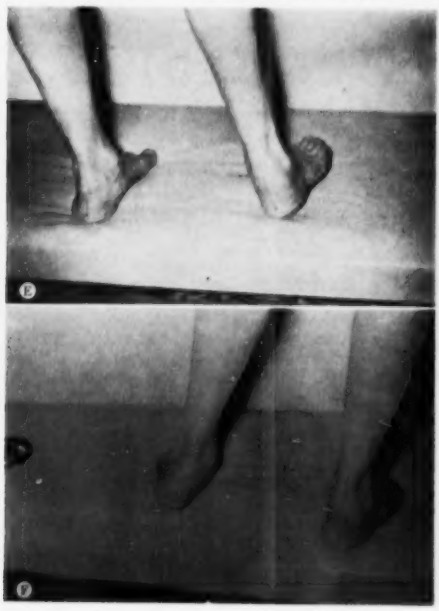
valgus foot which sometimes resulted after the Boehler method of treatment is no longer present. The fibrous fusion which apparently occurs in the subastragalar joint, as in the Boehler method, gives a painless foot in a shorter time. I have had no experience with early mobilization in "smash" fractures.

The long leg plaster applied after operation is left in place for six weeks. Then it is cut down into a short leg cast so that the patient may start knee motion. The patient wears this short leg cast an additional six weeks. Weight-bearing is not permitted until one thinks that the graft has revascularized sufficiently, as indicated by homogeneity of the trabecular pattern on roentgen examination. Revascularization is usually complete in 12 weeks, but the rate depends on whether cancellous or cortical bone has been used. If a longer time seems necessary, the patient carries out non-weight-bearing mobilizing activities during the waiting period.

I have not found it necessary to perform triple arthrodesis after the use of this meth-

Fig. 7C-D.—Inversion, right and left.

Fig. 7E-F.—Eversion, right and left.



Data in Seventeen Cases of Fracture of OS Calcis

Case No.	Sex	Age	Occupation	Workman's Compensation	Time of Return to Previous Occupation	Permanent Disability	Follow-Up Period	Comment
1	M	28	"Roughneck"	Yes	9 mo.	20% each foot	1 yr.	Bilateral
2	M	40	Locomotive engineer	Yes	4 mo.	5% of foot	14 mo.	Associated carpal scaphoid and Colles' fracture
3	M	35	Carpenter	No	6 mo.	20% of foot	7 mo.	
4	M	55	Boilermaker helper	Yes	6 mo.	15% of foot	1 yr.	
5	M	28	Farmer	No	6 mo.	Not rated; minimal limitation in eversion; ¼ in. calf atrophy		Associated fracture of multiple transverse processes
6	M	31	Carpenter	Yes	Undetermined	20% of foot		Sued employer for $185,000; associated fracture of pelvis and spine
7	M	54	Traffic manager	No	4 mo. on crutches; 6 mo. free-weight-bearing	Not rated, some discomfort when walked over 1 mile	9 mo.	Associated bimalleolar fracture, same side as fracture of os calcis; 1st- 2d- 3d-degree burns over right upper extremity and right shoulder
8	M	46	Ginner	Yes	Unknown	50% of body as a whole, due to spine injury	6 mo.	Severe compression fracture L-2 and L-5
9	M	43	Laborer	Yes	6 mo.	10% of foot	6 mo.	
10	M	49	Storekeeper for railroad	No	7 mo.	Limitation of subastragalar motion	1 yr.	Able to walk on concrete a full day
11	M	20	Lineman	Yes	Unknown	Unknown		Associated Colles' fracture; fracture of right fibula; a lawyer engaged in middle of treatment
12	M	40	Carpenter	Yes	4 mo.	15% of foot	4 yr.	
13	M	61	Carpenter	Yes	See "comment"	15% each foot	1 yr.	Has not returned to regular work as yet; returned to light work in 6 mo.
14	M	31	Service station operator	No	4 mo.	Not rated	2 yr.	
15	M	46	"Bracero"	Yes	5 mo.	Not rated	9 mo.	
16	F	46	Secretary	No				Under treatment
17	F	27	Housewife	No	8 mo.	Not rated	14 mo.	Associated fracture of tibia and D-10

od. The foot is more cosmetically acceptable than when early triple arthrodesis or delayed triple arthrodesis is performed for this same fracture.

Widén's analysis of 93 cases of fracture of the os calcis treated by open reduction points to favorable end-results.[18] My experience reveals a clinical impression which agrees with his conclusions (Table; Figs. 7A-B, 7C-D, 7E-F). With this method I now feel that I can, in most cases, return a man to heavy work within four to six months. It has not been necessary to perform triple arthrodesis or subastragalar fusion in any of the reported cases.[7,8] Although I have been using this method since June, 1948, my follow-up period is short because of difficulty in getting patients back for reexamination. However, I feel that my impression is valid, since the course in "smash" fractures is toward improvement rather than deterioration with the passage of time.[1,5,6]

Conclusions

1. No treatment of "smash" fractures of the os calcis is ideal.

2. Careful roentgen study of the posterior subastragalar joints permits a simple classification of fractures of the os calcis.

3. Residual symptoms from "smash" fractures diminish with the passage of time.

4. Open reduction modified after the method of Palmer has given me the best results.

Dr. Louis W. Breck and Dr. W. Compere Basom gave permission for the inclusion of their clinical data and cases in this paper.

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General Surgery

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The first question which arose in connection with the writing of this paper was "What comprises General Surgery?" More and more the content has been narrowed down to leavings after regional specialists have carved out areas. The titles of other papers in this symposium indicate that further divisions are being dispensed on the basis of specific tissues in whatever region they exist, such as cardiovascular surgery, or on the basis of the etiology of the surgical condition, in this case trauma. I like to think of both these subjects as being an integral part of general surgery, and the discussions actually on portions of the whole. This idea seems logical because advances in the field of cardiovascular surgery are exciting and accelerated, worthy of separate coverage. Advances in the surgery of trauma will come with dissemination of knowledge, which demands emphasis.

Another way of looking at the matter is to consider that general surgery is concerned with principles which apply to all surgical endeavor and with exploration into hitherto uncharted fields.

One of the most interesting of recent pursuits which has broad general implications began with a lesion called carcinoid, the very name being an attestation of ignorance. Like many other poorly understood disease processes, it is assigned to the surgeon. In 1954 [1] attention was called to a syndrome of cutaneous flushing, cyanosis, and valvular disease of the right heart which occurred in patients with metastatic carcinoid. The carcinoid tumors, metastatic deposits, and the blood of these patients contained large amounts of 5-hydroxytryptamine, or serotonin. Furthermore, a simple test for 5-hydroxytryptamine [2] in the urine is an excellent diagnostic sign of the presence of metastatic carcinoid. These slow-growing tumors have created many problems for surgeons. They arise from all parts of the gastrointestinal tract, and in a slow-motion pattern may spread to lymph nodes and liver, just as the more rapidly growing cancers do. A good diagnostic test for the metastases is therefore a valuable advance.

Further implications in studies of this disease lead to cardiovascular, endocrine, central nervous, and psychiatric considerations, all outside the bounds of general surgery. A brief consideration of a genuine "general" concept will not be out of place.

Carcinoid tumors were thought to have a connection with endocrine function as long ago as 1914,[3] when they were described as "Endocrine Tumors of the Appendix."

Two parallel investigations which bear upon the present concepts have been proceeding for years. One, physiologic and pharmacologic, started in 1918, with the discovery of a material recoverable from clotted blood that is vasopressor.[4] This material and its relation to hypertension and other vasoconstrictor phenomena have been studied extensively. It is called serotonin and is known to be 5-hydroxytryptamine, a metabolite of tryptophan. The other, parallel study, from 1944 on, revealed that the chromaffin cells in the gastrointestinal tract secreted an active hormone which was called enteramine [5] and was also shown to be a

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5-hydroxytryptamine, identical with serotonin.

The final meeting of these two lines has led to a divergence to other general fields, which will be mentioned here only to illustrate the artificiality of categorizing knowledge into subjects. Serotonin is known to enter into metabolic processes in the brain. It has been suggested that an abnormality of serotonin chemistry may be related to schizophrenia. Serotonin levels in the blood are markedly decreased by the administration of reserpine.[6] This line is leading too far into the subject of psychiatry for our present purposes.

Some further hypotheses have been proposed which are closer to general surgery. Excessive serotonin blood levels in metastatic carcinoid are associated with endocardial fibrosis in the right heart. Only in the presence of a shunt between chambers, such as occurs in patent foramen ovale, are both right and left heart lesions observed. Because of this unusual circumstance, it has been suggested that serotonin is inactivated in the lungs and reaches the left heart in sufficient concentrations to affect the left valves when blood is shunted.

A most intriguing proposal, that pulmonary arteriovenous fistulae are the shunts through which 5-hydroxytryptamine might escape detoxification in the lungs, has been made.[7] Over a sufficient period of time the effect of activated serotonin in peripheral blood in such an instance might be the explanation for so-called congenital telangiectasis, the Rendu-Osler-Weber syndrome. The high incidence of telangiectasis in pulmonary arteriovenous fistulae would support this contention.

A somewhat lengthy consideration of this advancing front of new knowledge has been presented to illustrate the lack of boundaries in investigation and the unpredictable directions from which information comes or toward which it leads.

Further significant recent progress in areas more closely concerned with a surgeon's activities will be summarized under various headings.

The subject of trauma is beyond the scope of this paper, but a broad study of injury in battle was conducted during the Korean War which should be saluted. This was a signal step, for it is the first time an organized and prepared team has been set up on the front lines for the specific purpose of studying wounded soldiers and recording detailed metabolic and physiologic data.[8] Previous research on the severely wounded in wartime had been conducted or organized by individuals, whose sense of inquiry pervaded their every action. In many instances the obstacles created by lack of Army support were insurmountable. These Korean War studies represent an advance in official policy and attitude toward investigation which is itself a milestone. The cooperation of engineers and supply and transportation branches enabled the research team to set up laboratories for complicated determinations, and to have skilled technicians at hand for immediate study of the wounded. Expansion of the team effort resulted in a controlled evacuation of wounded with uninterrupted observation throughout the soldiers' period under care. It is impressive to read in the research team's reports the emphasis placed on such studies as adrenal function, electrolyte changes, autonomic responses, and hepatic and renal function. The data are concerned with severe injury in a controlled group of healthy, well-nourished young men. To collect material of equal validity in civilian practice through fixed special laboratories would be an interminable task. Volume IV of the reports, on Post-Traumatic Renal Insufficiency, is especially recommended as typical of this great contribution.

To continue with important subjects of general interest and of a general nature, progress in the field of tissue transplantation is being made. Ability to replace worn-out organs by transplantation from one individual to another or from one species to another will probably be the next great application of surgical skill. The mystery of how an organism recognizes transplanted alien skin (to consider the most conveniently transplantable tissue) only after

host blood has circulated through it, and then promptly rejects the foreign skin is unsolved. Permanent survival of homografted skin occurs in the very closest of genetic links—monozygotic twins—and probably in congenital agammaglobulinemia. This principle is true of all other tissues, even though cornea, bone, etc., are slowly rejected and replaced by host tissues. Absence of gammaglobulin is not alone the factor which permits homotransplantation, because a child with acquired agammaglobulinemia rejects homografts, although later than a normal child. Investigations reported at the Second Tissue Homotransplantation Conference [9] are pursuing numerous clues. Among these are prolonged survival time, which may be induced by cortisone; the receptivity for homografts in embryos or the newborn in some species; a genetic link short of that in monozygotic twins which prolongs survival time, and, most exciting, the detection of a principle in cell transfers which may be the antibody mechanism.

The most rapidly advancing front of applied general surgery is that concerned with infants and children. A group of pioneer surgeons who begin with a knowledge of metabolism in a small infant, its needs and limitations, have brought about a new attitude toward the most complex of congenital anomalies.

There are several major fronts in the attack on such congenital anomalies and diseases of the newborn. Swenson's [10] important elucidation of the mechanism of congenital megacolon, i. e., absence of ganglion (?) cells in a segment of colon which in effect produces an obstructive element is a starting point of one of these investigations. Resection of the whole of the aganglionic segment corrects the disorder, and results after 10 years have been reported to be good. In other areas of the gastrointestinal tract, similar absence or immaturity of ganglia has been demonstrated—for example, in the lower esophagus and in the gastric pylorus, producing achalasia and congenital pyloric stenosis, respectively (Friesen [11]).

Proper understanding of the metabolic peculiarities of infants has helped enormously in bold surgical treatment of atresias and stenoses of the bowel, meconium ileus, and tracheoesophageal fistulae. The reduced mortality of operations in these conditions is an outstanding achievement.

In other advances which involve liver disease, ordinarily considered a medical condition, surgical implications are interwoven. Hepatic coma which complicates cirrhosis of the liver has been attributed to elevated blood ammonia levels. Ammonia in portal vein blood arises from nitrogenous materials in the gastrointestinal tract. The normal liver synthesizes ammonia to urea, which is excreted by the kidney. In patients with cirrhosis this ability may be impaired. In the Eck-fistula dog (portal vein anastomosed to inferior vena cava) meat intoxication was known to Pavlov. Patients in whom an Eck fistula is created surgically for relief of portal hypertension are subjects for so-called meat intoxication or ammonia poisoning. Some neurological episodes, stupor and mental confusion, which occur in patients with portacaval shunts performed for the treatment of esophageal varices, have been attributed to this same mechanism.[12] Some questions have been raised regarding the precise role of ammonia in the blood in these episodes and in hepatic coma, for correlation between blood ammonia levels and symptoms has not been perfect.[13] Hepatic coma has been observed in the presence of normal blood ammonia, and elevated blood ammonia is not necessarily accompanied by symptoms. In any case the original concept of meat intoxication holds, whatever the precise mechanism. This is an important point because blood in the gastrointestinal tract is strong meat. In a patient with cirrhosis, whether or not a previous shunt has been performed, bleeding from esophageal varices must be treated with this in mind. Not only should the bleeding be stopped by some means, but also saline catharsis, administration of antiobiotics, and irrigations to remove

the blood from the gastrointestinal tract should be instituted.

The whole field of technical surgery has advanced, along with knowledge of basic metabolic responses to operation. Students of endocrine function, anesthesiologists, and biochemists have contributed to an extent which has decreased the risk of prolonged and extensive operations progressively.

Among the purely technical steps which have contributed to the comfort and welfare of patients is decompression of the gastrointestinal tract in the postoperative period by a gastrostomy tube rather than by an indwelling nasogastric tube.[14] I know of no simple method which has provided so much benefit. On one hand, the desirability of continuous decompression of the stomach and upper gastrointestinal tract after some operations is unquestioned. On the other, the complications arising from indwelling tubes must be weighed. Mucus secretion in the pharynx and trachea with suppressed cough can lead to pulmonary atelectasis; reflux of gastric juice through the distorted esophagogastric junction can result in esophagitis, and even severe erosions of the pharynx and esophagus are not infrequent. The gastrostomy is made through a stab wound, with a few sutures attaching the stomach to the anterior abdominal wall. When the decompression is no longer required, removal of the tube is followed by prompt closure of the wound.

Brief mention should be made of several other proposals in general surgical management which are for time to evaluate.

An ingenious method of promoting absorption of ascitic fluid has been developed in dogs and applied to a small number of human patients.[15] The absorptive powers of the lower ileum are utilized by isolating and opening a pedicled loop of bowel and attaching it to the lateral abdominal wall, with the mucosal side exposed to the peritoneal cavity.

A study of tumor cells in the blood stream has been conducted[16] which reveals that in both operable and inoperable cases such cells are found in a large percentage. There seems to be no precise correlation of manipulation of the tumor, extent of metastases, survival time, and the number of cells so demonstrated.

Splenectomy for hypoplasia of the bone marrow, aplastic anemia, has been found to be of moderate benefit[17] in certain instances of this dreadful disease to a degree that the patient is enabled to exist without dependency on transfused blood.

Such an imposing task as summarizing recent advances in General Surgery can be little more than the experiences impressed on one person. I have tried to point out some of those items which seem to be of special significance to me. Undoubtedly, many important advances have been omitted either because I missed them or because I did not appreciate their real value. It is true that a principle may be old and established, but it is new to the person hearing of it for the first time. Reading John Hunter's 200-year-old works can give the thrill and excitement of something new!

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Technique for Collection of Sterile Bile from Experimental Animals

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Difficulty in obtaining large quantities of sterile bile has led to the development of several techniques in various animal species for prolonged collection of bile. The continuing sterility of these methods is open to serious question.

In the rat, which has no gallbladder, bile has been collected by cannulating the common duct and allowing bile to drain into specially constructed glass bulbs which were placed either in the peritoneal cavity [1,2] or on the abdominal wall.[3] Bile was withdrawn at specified intervals from the glass bulbs through rubber tubes. Another technique used a long plastic tube extending from the common duct through a subcutaneous tunnel to the dorsal surface of the rat's foot to permit continuous collection of bile while the animal was moving about.[4]

In the rabbit the common duct has been divided between ligatures, and a flexible tube inserted into the gallbladder and brought to the outside to a collecting rubber bag.[5]

In the dog the technique of Rous and McMaster has been the usually accepted procedure.[6] This consists of common-duct intubation after common- and cystic-duct ligation, and collection of bile in a rubber balloon on the abdominal wall. Two catheters in the common duct were utilized in other methods, which permitted collection of bile, or reintroduction of bile into the duodenum through selective use of the two catheters.[7,8] The gallbladder has been utilized in conjunction with common-duct ligation in other techniques, which either had the gallbladder cannulated just beneath the skin [9] or else had a gold-plated cannula inserted in the gallbladder and connected to a collecting balloon on the outside.[10,11] More complicated procedures placed the duodenum subcutaneously so that at a subsequent operation the common duct could be isolated, divided, and allowed to drain through a catheter inserted at will [12] or the duodenum could be opened and bile allowed to flow freely on the abdominal wall.[13]

In each of these techniques there are multiple opportunities for contamination of the bile, since there are several connecting links in each system, and also since most methods used some external type of collecting system in which the bile could be stored until collected.

Methods

The basic principles of the technique to be described are that the animal's own collecting system is utilized, there is only a single opening into the biliary system through which contamination might occur, and there is no fixation between gallbladder and other viscera to serve as a further cause of contamination.

Dogs are operated upon under intravenous pentobarbital (Nembutal) anesthesia. Sterile techniques are used throughout. A high right rectus incision is made. The common duct is isolated below the lowest hepatic duct, since these ducts enter below the cystic duct in the dog (Figure). The duct is doubly ligated and divided to prevent later recanalization of the duct. Two purse-string sutures of 000 silk are placed in the fundus of the gallbladder, and through an opening in the center of the sutures is inserted a No. 15 polyvinyl plastic tube,* which has been sterilized by autoclaving. The tube is threaded through the omentum and the omentum

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* Obtained from Pharmaseal Laboratories, Inc.

Drawing of operative procedure. Common duct ligated and divided below lowest hepatic duct and close to duodenum. Plastic tube inserted in fundus of gallbladder and held in place with purse-string suture.

sutured to the fundus of the gallbladder. The plastic tube should be 12 to 18 in. long, so that sufficient tubing can be left within the abdomen to permit normal motion of the viscera without exerting undue traction on the tube.

The tube is exteriorized through a stab wound. A short, blunt No. 15-gauge needle is inserted in the tube and a metal adapter plug applied. The exteriorized portion of the collecting system is fixed to the abdominal wall with sterile dressings and tape and the entire area enclosed in a snug abdominal binder, so that the dog cannot get at the collecting apparatus.

The animals receive 600,000 units of procaine penicillin G twice daily from the time of surgery until bile is no longer desired. Bile salts, 0.6 gm. three times a day, are administered orally to stimulate bile production and also to prevent too severe depletion of bile salts and fat-soluble foods.

Bacterial Studies on Bile

Dog No.	No. Days Bile Sterile	Contaminating Organism
7	3	A. aerogenes
17	5	Pseudomonas sp.
27	16	None
33	5	Pseudomonas sp.
44	5	A. aerogenes
55	10	Pseudomonas sp.
85	10	None
96	15	Pseudomonas sp.
97	18	Pseudomonas sp.
1000	10	Pseudomonas sp.

Bile is collected three times daily by aspirating with a fresh, sterile Luer-Lok syringe and immediately transferring the bile to a sterile flask for storage under refrigeration. While each specimen is being withdrawn, the metal adapter plug is immersed in acid-alcohol and the tip of the adapter is wiped with the same solution to prevent contamination during withdrawal of the specimens.

Each specimen is cultured aerobically and anaerobically. Penicillinase has been added to the culture medium, since the penicillin level in the bile has been found to be high enough to inhibit, at least theoretically, some growth in the collected specimens. When contaminated specimens are found, they are recultured without penicillinase to see whether such cultures would have given a false sense of security.

Results

Seventeen dogs were subjected to this procedure, and bile collected from ten of these animals remained sterile for 3 to 18 days (Table). The median duration of sterile-bile production was 10 days.

Seven dogs were discarded. One never produced bile; three had contamination from the start, and in three dogs the tube came out of the gallbladder in the immediate postoperative period.

After the first 72 hours, the daily output of bile varied between 3 and 100 ml. Bile production was limited during the immediate postoperative period. The total amount of bile collected from these animals ranged from 75 to 1471 ml. The animals all remained active and alert and had a good

appetite as long as the collecting apparatus was intact and the bile remained sterile.

Cultures showed that the main contaminants were Aerobacter aerogenes and Pseudomonas sp. Bile collection was discontinued whenever the presence of organisms was confirmed in successive specimens. Two do not show contamination in the Table because collection of bile was discontinued when the needle came off the plastic tube and it was felt that contamination could not be avoided. The source of the contaminating organisms is not known, but the type organisms found suggests that the contamination was in some way related to the collecting system rather than to primary contamination of the bile.

Summary

A technique is described for the repeated collection of sterile bile which utilizes the animal's own collecting, concentrating, and storage apparatuses for their normal functions. The technique cannot be used to collect hepatic bile.

The opportunities for infection are reduced by eliminating complicated collecting systems, and by eliminating sutures between the biliary system and other viscera.

Bile so obtained may be used to study any of the properties of bile or the excretion of various substances in bile without the interference of contaminating bacteria.

Large quantities of normal, sterile gallbladder bile are thus available for any study desired.

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Stenosis of the Stomach Following Ingestion of Lye

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Introduction

Whenever corrosive substances are ingested, both the esophagus and the stomach may become the site of stenosis. Acids, in general, destroy primarily the stomach, while alkalis primarily damage the esophagus, as the latter agents are usually neutralized by the acid contents of the stomach. When stenosis results, it usually occurs in narrower areas of the esophagus, where there may have been momentary "hold up" of the ingested material. In the stomach, pyloric stenosis is most apt to occur.

Before 1948 only 139 cases of pyloric obstruction following ingestion of a corrosive substance had been recorded; 11 of these were reported by American authors.[1,3,8,11,12] Since 1948 a few additional cases have been reported.[2,7] Controversy remains regarding the best surgical therapy for those cases in which the stomach becomes stenotic.

Historical Review

The first report of a case of pyloric stenosis following ingestion of corrosive substances was made by Nobert[9] in 1828. In his case, sulfuric acid was the solution ingested. Since then, the most commonly reported corrosive agents producing pyloric obstruction have been acids. Heindl,[5] however, reviewed 116 cases of lye strictures of the esophagus, in 20% of which pyloric stenosis was present. Gray and Holmes[4] found an additional 10 cases showing stenosis of the esophagus and the pylorus and 4 cases showing stenosis of the pylorus with no damage to the esophagus. The surgical treatment of pyloric stenosis caused by the action of ingested corrosive substances has consisted of (1) jejunostomy, (2) digital dilation of the stricture, (3) pyloroplasty, (4) gastroenterostomy, and (5) partial gastrectomy.

A feeding jejunostomy has been, and is still, recommended for those early cases in which the reaction is so severe that operations upon the stomach are hazardous. It is meant only as a temporary, life-saving measure.

Loreta,[6] in 1884, was apparently the first to describe an operation designed as definitive therapy. He advised digital dilation of the pyloric stricture, but found, as did White and Lane, that the stenosis was almost certain to recur. Consequently, this procedure has been abandoned.

Senn,[10] followed by others, advocated pyloroplasty. The disadvantage of this has been that the area of stenosis, in many instances, was so extensive that operation was of no benefit, or impossible to perform.

The majority of surgeons treating gastric strictures have performed a gastroenterostomy. Gray and Holmes[4] felt that this operation had several distinct disadvantages, namely, (1) that the anastomosis had to be performed in an area of the stomach also partially diseased, and therefore was a risk to the patient, (2) that a gastric or marginal ulcer might develop, and (3) that neoplastic change might occur in the diseased portion of the stomach. However, partial gastrectomy, strongly advocated by Gray and Holmes, has been employed only a few times.

Report of Cases

CASE 1.—A 67-year-old white woman drank a spoonful of diluted lye (sodium hydroxide) in a suicide attempt on July 24, 1955. One hour later she was subjected to gastric lavage with weak acetic

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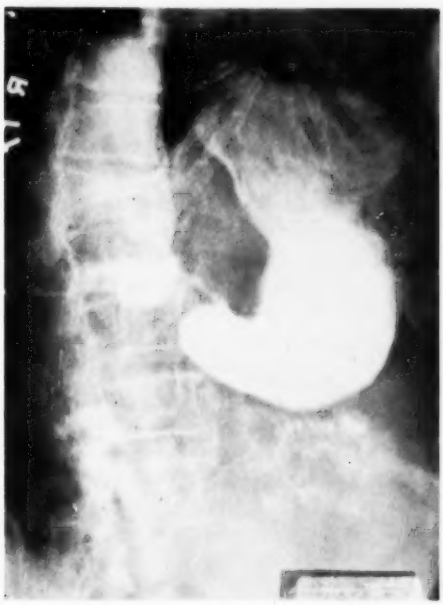
acid solution in the emergency room of another hospital. Her early symptoms were only burning of the mouth and throat. For one week she was fed parenterally. Because of a temperature of 102 F and a white blood cell count of 25,000 per cubic millimeter, she was given tetracycline (Achromycin).

She was then referred to Illinois Research and Educational Hospital on Aug. 4, 1955, where examination revealed a pale, elderly-appearing white woman, able to sit up but too weak to walk. Her upper and lower lips, dorsum of tongue, soft palate, lateral and posterior walls of pharynx, and epiglottis were red, swollen, partly ulcerated, and lined by necrotic material. The vocal cords were only slightly injected, and there was no airway impairment or voice change.

An esophagogram on the day of admission revealed a normal swallowing mechanism and a normal-appearing esophagus. She was started on daily esophageal dilations, beginning with a No. 24 Hurst mercury bougie, which passed easily into the stomach. By Aug. 21 a No. 40 mercury bougie was passed with ease into the stomach, and the patient was discharged from the hospital on a soft diet. Dilations were continued on an outpatient basis.

On Oct. 7 the patient was readmitted because she was no longer able to retain anything but liquids taken orally and complained of epigastric burning and a noticeable decrease in the number of bowel movements. She was tried on rigorous ulcer management, without relief. An upper gastrointestinal x-ray study revealed a prepyloric stenosis (Fig. 1). Gastric analysis revealed 10 mEq/L. of free acid and 64 mEq/L. of total acid.

Fig. 1.—Stomach roentgenograms in Case 1, showing prepyloric lye stricture with dilated stomach.



On Nov. 15 the patient's abdomen was explored through a high left paramedian incision. A prepyloric stenosis, measuring about 2 in. (5 cm.) in length, was observed. A posterior gastroenterotomy was then performed.

Postoperatively, the patient did exceptionally well and was discharged on a full diet eight days later. She was last seen on April 9, 1957, at which time she had gained 25 lb. (11.3 kg.) and was eating a normal diet.

This case revealed only moderate esophagitis and esophageal stricture but a severe prepyloric stenosis of the stomach. Because of her relatively low gastric acid values, it is hoped that she will not develop a gastric or stomal ulcer. A subtotal gastrectomy seemed to carry a prohibitive risk because of the patient's severe malnutrition and dehydration. A simple gastroenterostomy quickly restored her to normal alimentation and health. Eighteen months after the surgery the patient was asymptomatic.

Case 2.—A 21-year-old white woman drank about one-half glass of lye (sodium hydroxide) in a suicide attempt 10 weeks prior to admission. She had received emergency treatment (lavage with milk and eggs) 10 minutes after ingestion. After emergency treatment, she was given an oral liquid diet, later supplemented by intravenous alimentation. During the following 10 weeks the patient received frequent esophageal dilations. In spite of this, she could swallow only liquids, frequently vomited, and lost 40 lb. (18 kg.) in weight.

When she was admitted to Illinois Research and Educational Hospitals, there were marked symptoms and signs of dehydration and malnutrition. An esophagogram revealed severe stricture of the entire esophagus. Esophagoscopy revealed moderately severe esophagitis. Daily dilations of the esophagus were done, and parenteral feedings, antibiotics, and prednisone (Meticortin) were administered.

After two weeks of such management, the patient was still unable to retain anything taken orally, even though her esophageal lumen was considered adequate. Believing the cause to be gastritis, we placed the patient on ulcer management, without relief. An upper gastrointestinal x-ray study at this time revealed a complete stricture high in the stomach (Fig. 2). By this time she had lost 70 lb. (31.5 kg.) in weight.

On Nov. 14, 1955, after maximum improvement in fluid and electrolyte balance had been obtained, a

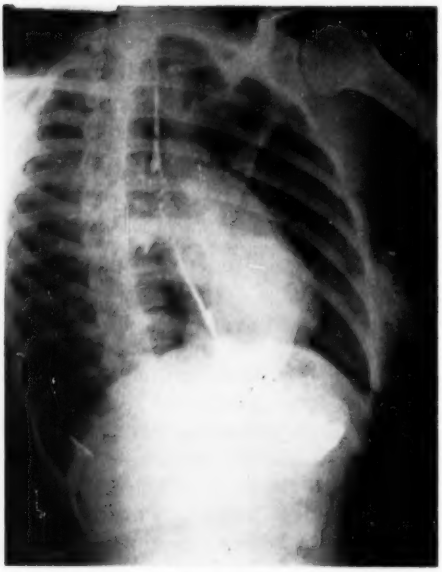


Fig. 2.—Stomach roentgenogram in Case 2, showing high and complete stenosis due to ingestion of lye. The marked esophageal stricture is also shown.

celiotomy was carried out. The entire midportion of the stomach was completely stenotic. The prepyloric area was apparently uninvolved. A small dilated gastric pouch remained high in the cardia. A second, incomplete stricture partially divided this pouch into two dilated areas.

A posterior gastroenterostomy was performed high in the cardia, distal to the partially stenosing band. The patient made an uneventful recovery. She was eating a soft diet without difficulty one week later and was discharged on her 26th postoperative day.

Esophageal dilations were continued on an outpatient basis. Soon, however, the patient again began to vomit. A repeat upper gastrointestinal x-ray with thin barium revealed a nonfunctioning gastroenterostomy, and she was readmitted to the surgical service. At this time the esophagoscopist reported that he was no longer able to dilate the esophagus beyond the level of the aortic arch.

It was now obvious that the patient would require almost total reconstruction of an esophagus with by-pass of her stomach, or a feeding jejunostomy. It was decided to reconstruct the esophagus; so on Jan. 9, 1956, a presternal esophagus was reconstructed from the right hemicolon, with the distal portion anastomosed to the duodenum and the cecum brought out in her neck as a cervical cecostomy (Fig. 3). She tolerated the operation well and was soon tube-fed through the cecostomy. Cervical esophageal dilatations were continued.

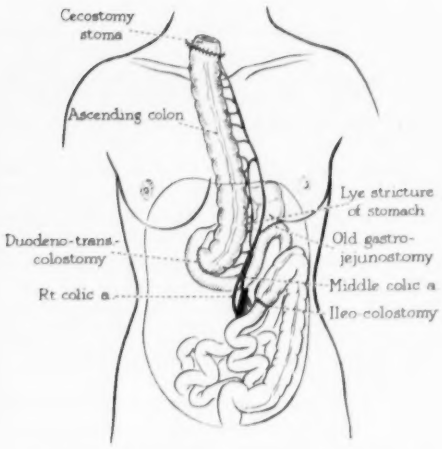


Fig. 3.—Schematic representation of construction and anastomoses in the presternal colon transplant used in Case 2.

On Feb. 2 the cervical esophagus was anastomosed to the cecum in the neck. Seven days later the patient was started on an oral diet, ate a steak sandwich the following day, and was discharged. Cervical esophageal dilations have been continued on an outpatient basis.

She was last seen Dec. 28, 1956, at which time she had gained back 35 lb. (15.8 kg.) weight and was eating a regular diet.

This patient drank lye in a suicide attempt, with resultant total severe stricture of the esophagus, as well as nearly total stricture of the stomach. A high posterior gastroenterostomy was attempted first in an effort to restore her to normal alimentation. This failed to function for long, however, and soon the lower esophagus resisted all attempts at dilation. Consequently, the esophagus was reconstructed, using the right hemicolon brought up to the cervical esophagus in a presternal position and anastomosed to the duodenum. Dilations of the remaining cervical esophagus have been continued. No ill effects have resulted from the stenosed thoracic esophagus and stomach, which have been left in situ and by-passed. The patient is now gaining weight and is able to eat a regular diet.

Case 3.—A 47-year-old white woman attempted suicide July 15, 1955, by taking 2 spoonsful of lye in a glass of water. She was taken to a local hospital, where her stomach was lavaged with egg

white and milk. After this, she was kept on a liquid diet for three weeks, but, because of increasing difficulty in swallowing, she was transferred to Illinois Research and Educational Hospitals on Aug. 24, 1955. An esophagogram the day of admission revealed stricture in the midesophagus 6 cm. in length, with the upper limit at the carina. Esophagoscopy the same day revealed moderate to severe esophagitis and a stricture at 31 cm. The esophagus was then dilated with bougies up to a No. 26 F. with ease.

Esophageal dilations were carried out at three-week intervals in the outpatient department. Although the dilations were performed with ease, the patient soon began to vomit her feedings and rather rapidly lost 50 lb. (22.5 kg.) in weight.

Because of her poor clinical course, she was readmitted to the hospital Dec. 13, 1955, at which time she weighed only 84 lb. (37.8 kg.). Esophagoscopy revealed moderate esophagitis and a stricture at 31 cm., which was passed with ease. An upper gastrointestinal x-ray, using thin barium, showed a stricture of the midportion of the stomach (Fig. 4).

Abdominal exploration was carried out on Dec. 22. Examination of the stomach revealed a dilated proximal segment consisting of the fundus and the upper one-third of the body. The remaining two-thirds of the body and the pyloric vestibule were stenosed, with a thickened, firm wall. By palpation, it was impossible to differentiate this from a gastric neoplasm of the linitis plastica type. There were many enlarged, rubbery, firm lymph nodes in the adjacent gastrocolic ligament. Several of these were removed for frozen-section biopsy and demonstrated only lymphoid hyperplasia. The pyloric antrum and pylorus were normal.

The proximal, dilated stomach was then opened posteriorly in its most dependent portion. The pouch was explored with a palpating finger. The cardiac antrum was open and apparently not diseased. The strictured portion of the stomach contained a channel so small that it would not admit the end of a Péan forceps. Even the wall of the nonstenosed portion of stomach was thicker and firmer than normal. A routine high retrocolic posterior gastroenterostomy was performed.

Postoperatively the patient made an uneventful recovery. The day following surgery feeding was begun through a Rawson-Abbott tube, using high-protein, high-caloric substances. The tube was removed on the fourth postoperative day. The patient soon tolerated a general hospital diet well. Ten days after surgery the patient had gained 10 lb. (4.5 kg.) in weight and was discharged. She was last seen April 25, 1956, at which time she had gained 28 lb. (12.6 kg.), was eating a normal diet, and was asymptomatic.

Fig. 4.—Gastric roentgenogram in Case 3, showing lye stricture of midportion of stomach.

This patient drank lye in a suicide attempt, with resultant marked esophageal stricture and stricture of the midportion of the stomach. By the time the stricture of the stomach was recognized, she was in such a severe state of malnutrition and dehydration that partial gastrectomy seemed unduly hazardous and a gastroenterostomy was performed. At the time of the surgery, it was impossible to differentiate the diseased portion of the stomach from a linitis plastica type of gastric carcinoma by palpation. Four months later the patient was asymptomatic, was eating a normal diet, and had regained 28 lb.

Summary

Three cases of lye strictures are presented showing three different pathologic pictures: (1) marked prepyloric obstruction of the stomach with minimal stricture of the esophagus; (2) almost complete stricture of the esophagus and stomach, and (3) marked stenosis of the midportion of the stomach with equally marked esophageal stricture

In all three cases the stricture of the stomach manifested itself much later than in the esophagus. This is to be expected because of the great differences in the size of the lumina.

In all three cases the stricture of the stomach was undiagnosed immediately and was mistaken for gastritis.

Gastroenterostomy was attempted initially in all three cases, as it seemed the quickest and safest way to restore them to normal alimentation. The general state of cachexia and dehydration exhibited in all three cases made partial gastrectomy seem unduly hazardous.

One case eventually required almost complete reconstruction of the esophagus with by-pass of the stomach. This was done with a presternal transplant of the right hemicolon, and it has proved a satisfactory solution to date.

Whenever corrosive substances are ingested, obstruction of the stomach, as well as stricture of the esophagus, may occur. Examination of these patients should include an upper gastrointestinal barium x-ray study, as well as esophograms. The surgical treatment of strictures of the stomach secondary to the action of corrosive substances has not been standardized. The reporting of long-term follow-up studies of various types of surgical therapy may establish a standard form of therapy, as well as indicate delayed complications which may be expected.

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Studies on Hypothermia in Abdominal Surgery

II. Occlusion of the Vascular Inflow to the Liver

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The successful application of hypothermia to the surgery of the heart and brain is too well known to call for extensive documentation. Our own studies, which began with the extension of the safe time of occlusion of the abdominal aorta by means of hypothermia, reported in Part I,[1] have been extended to the specific devascularization of the liver. These latter experiments will be reported here.

Resections of the liver in normothermic patients have been successfully accomplished by several surgeons. The largest series appears to be the four patients reported by Pack and colleagues.[2] The hazard of extensive blood loss, and of serious bile leakage owing to failure of complete bile-duct ligature in a sanguineous field, have kept these operations from gaining general acceptance. Bernhard and colleagues[3] have reported experiments similar to ours which indicate that a significant increase in safe operating time may be achieved by the use of hypothermia in combination with appropriate vascular occlusion.

Temporary occlusion of the hepatic artery and portal vein in an effort to obtain a bloodless liver was first reported by Duchinova[4] in 1925. Fatalities occurred after only a few minutes of occlusion, varying up to 30 minutes occasionally, but in general after even shorter periods. The shock-like death which ensues has been extensively studied, most recently by State and Lichtenstein,[5] but is still imperfectly understood. Suggested etiologic factors include (*a*) sequestration of blood in the portal system[6]; (*b*) elaboration of VDM (vasodepressor material) by the damaged liver[7]; (*c*) production of a bacterial factor,[8,9] and (*d*) decrease in effective circulating blood volume by hemorrhage into the liver from the hepatic artery.[5]

With this background, we felt that if the vascular inflow to the portal bed was occluded in addition to the portal vein and hepatic artery, the element of sequestration of blood in the portal system would be minimized. We felt, furthermore, that if general body cooling was employed for its depressant effect upon metabolism, some of the deleterious results of hepatic anoxia might be postponed for a useful period of time.

Experiments

Mongrel dogs in good health and ranging in weight from 10 to 25 kg. were used. No specific dietary preparation was employed.

Preliminary observations disclosed that application of a clamp to the porta hepatis, including the portal vein, hepatic artery, and common bile duct, led to prompt edema and congestion of the small bowel and of the proximal 3 to 4 in. of the large bowel. When occlusion of the superior mesenteric artery was added, edema of the bowel was greatly reduced, but the spleen became markedly enlarged. If occlusion of the splenic artery was added to the previous two occlusions, the spleen remained normal in size, or was distended to a slight degree. Originally, these three occlusions were employed until we found that it was more satisfactory to occlude the whole celiac axis instead of simply the splenic artery. This prevented gastric engorgement, which was found to have occurred during prolonged occlusion if the left gastric artery was patent. It was found to be unnecessary to clamp the inferior mesenteric artery. Our standard oc-

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clusion, therefore, consisted of applying clamps to the following three vessels: (1) superior mesenteric artery, (2) celiac axis, and (3) porta hepatis.

Three groups of animals were studied, as follows:

1. A control series, in which the standard occlusions described above were applied to normothermic dogs, in order to determine the maximum safe time of occlusion.

2. A hypothermic series, in which the same procedure was followed on animals cooled to 28 C.

3. A hypothermic-operative series, in which animals were cooled and the occlusions maintained for 60 minutes, during which time the right lobes of the liver and the gallbladder were resected. The purpose of this group was to determine the ability of the animals to withstand extensive liver resection under these conditions, and also to test the ability of the liver remnant to survive and continue its function.

Methods

1. *The Control Series.*—All animals in the control series were subjected to the following routine.

The animals were brought down from the animal house to a quiet room one hour prior to operation and given an intramuscular injection of meperidine (Demerol). The amount of meperidine given depended on the body weight of the animal calculated from a pediatric scale. The usual dose was 18-20 mg. They were also given 600,000 units of penicillin at this time to counteract the bacteremia which has been shown to follow occlusion of the hepatic artery.[8]

After an interval of one hour, during which time the meperidine had made the animal quite drowsy, anesthesia was induced, using an intravenous solution of pentobarbital sodium (Nembutal). The pentobarbital sodium solution was in a concentration of 1 grain (65 mg.) per cubic centimeter and the amount used was 1 cc. per 5 lb. body weight. This produced very good surgical anesthesia for at least three hours. An intravenous infusion of 5% dextrose and water was started at this time to counteract the hypoglycemia which might have resulted when the blood supply to the liver was occluded. The abdomen, thorax, and neck were shaved, prepped and draped in accordance with strict aseptic technique.

Blood pressure recordings were taken on all animals. A midline incision was made in the neck below the level of the thyroid cartilage, and the right common carotid artery was isolated. A cannula was inserted in the artery and connected to a mercury kymograph.

A midline abdominal incision, extending from the xiphoid to the umbilicus, was employed. Skin towels were applied to the skin edges, and the peritoneum was opened. Large self-retaining retractors were inserted and spread completely to afford excellent exposure. The porta hepatis was isolated by passing a finger in the foramen of Winslow and out through the lesser omentum. A tape was placed around the porta hepatis for future identification. Palpation of the hepatic artery was always performed to assure its inclusion in the structures isolated by the tape. The spleen and bowel were then packed off to the right by means of abdominal towels. The celiac axis and superior mesenteric artery were identified and dissected free at their origins from the aorta, with the splenic vein crossing the aorta between these vessels and the left renal vein crossing the aorta below the superior mesenteric artery. Identifying tapes were passed around these arteries. If the splenic artery was to be occluded, as was done in the earlier part of the series, it was identified at its origin from the celiac axis. The vessels were occluded with bulldog clamps. The superior mesenteric artery was occluded first, then the celiac axis, and lastly the porta hepatis. The vessels were occluded in this order so that the portal pressure was not raised. After the required period of occlusion, the clamps were removed in the reverse order, that is, the porta hepatis first, then the celiac axis, and lastly the superior mesenteric artery. The abdomen was closed in layers. The cannula was removed from the right common carotid artery and the distal end of the artery doubly ligated. The neck was closed and the dog returned to the animal house.

2. *The Hypothermic Series.*—In the hypothermic series exactly the same routine was followed concerning premedication, antibiotic therapy, intravenous therapy, blood pressure recording, preparation for operation, and operative procedure as was used in the control series. Hypothermia was induced to 28 C by means of a lytic cocktail and external cooling, according to the following technique.

One hour following premedication the dog was anesthetized with an intravenous injection of thiopental sodium (Pentothal sodium) in a concentration of 1 grain per cubic centimeter. The usual initial dose was 3-5 cc. An intravenous infusion of 5% dextrose and water was started, with a stopcock for further intravenous injections of thiopental and the cocktail. A rectal thermometer was placed in the rectum for a distance of 2 in., and the temperature was recorded. It was left in place for future temperature readings. Ice cubes placed in polyethylene bags were applied to the groins, abdomen, and chest.

The animal was given 5 cc. of a lytic cocktail intravenously. This lytic cocktail contained chlorpromazine (Largactil) hydrochloride 25 mg., promethazine N. F. (Phanergan) hydrochloride

25 mg. and meperidine hydrochloride according to body weight. The amount of meperidine was determined from a pediatric scale, the average dose being 18-20 mg. These three drugs were diluted in 20 cc. of isotonic saline. A further 2 cc. of the lytic cocktail was given every 15 minutes until the rectal temperature fell to 29 C.

Further injections of 1 cc. of thiopental sodium were given whenever needed to control restlessness until the rectal temperature fell to 34 C. Below this temperature only 0.5 cc. doses were employed. In actual fact, once the temperature reached 32 C, further anesthesia was rarely needed. The average dog seldom received more than 7 cc. of the thiopental sodium solution during the whole procedure.

All dogs were intubated at 30 C as a precaution in case artificial respiration was needed. In actual fact, this was rarely required. Once the rectal temperature fell to 29 C, all cooling was stopped, as the temperature usually fell a further 1 to 2 degrees (C) after removal of the ice packs.

The animal was then taken to the operating room and positioned on its back on the operating table. One electric heating pad was placed under the back and another over the chest. This enabled rewarming to be begun as soon as the occluded vessels were released, while the abdomen was being closed. The operative procedure described above for the control series was then carried out. Postoperatively a baker was placed over the abdomen and, together with the two heating pads already in place, the dog was rewarmed at a rate of approximately 1 degree (C) every 15 minutes. The intravenous infusion was continued. The animal was extubated at 32 C and rewarming continued until 36 C, at which time the dog was wrapped in a blanket and returned to the animal house.

It was interesting to note that shivering, a frequently reported complication during the induction of hypothermia, was not encountered in a single case.

3. *The Hypothermic-Operative Series.*—In this series hypothermia was induced to 28 C in the manner just described. With the vascular supply to the liver occluded for 60 minutes, the animals were subjected to cholecystectomy and resection of the right lobes of the liver. One dog had a splenectomy performed as well.

A right subcostal incision was made 1 in. below the costal margin. The incision was carried down through the right rectus, external oblique, internal oblique, and transversus abdominis muscles. Occasionally it was necessary to extend the incision to include the left rectus muscle to obtain better exposure. The peritoneum was opened, and a large self-retaining retractor was inserted and spread completely to afford very good exposure of the right half of the liver. The superior mesenteric artery, celiac axis, and porta hepatis were isolated and occluded with bulldog clamps. A routine cholecystectomy was performed, with identification and ligation of the cystic artery and duct.

Resection of the right lobes of the liver was carried out in the following manner: In the first three dogs a suture was placed through the substance of one of the liver lobes so that the lobe could be moved in any direction with ease. Guiding the lobe with this suture, a Miller intestinal clamp was closed across the lobe as near to the inferior vena cava as possible. The lobe was then cut away. The remaining lobes of the right half of the liver were removed in a similar fashion. An attempt was made to oversew the cut edge of the liver, but this proved unsatisfactory, as the capsule of the devascularized liver in the dog will not hold a suture well. The method was also unsatisfactory, as the placing of the Miller clamp was a blind procedure and there was always the danger of damaging the inferior vena cava. It was noted during these resections that the only bleeding of any note came from the hepatic veins, which were easily identified and ligated with transfixion sutures. The rest of the cut surface of the liver remained surprisingly dry, after the initial blood was sponged away. This prompted the use of a more radical approach.

Accordingly, in the remaining animals in this series, the right lobes of the liver were dissected away from the hepatic veins by sharp and blunt dissection. The procedure was surprisingly easy, due to the practically bloodless operative field. In this way the hepatic veins could be ligated with transfixion sutures under direct vision very close to the inferior vena cava. This eliminated the more or less blind clamping of the liver lobes. It was found to be unnecessary to place absorbable gelatin sponge U. S. P. (Gelfoam) against the cut surface of the liver, even after the clamps were released from the porta hepatis, celiac axis, and superior mesenteric artery. The abdomen was closed in layers without drainage. Rewarming was continued to 36 C, and the animal was then returned to the animal house.

Results

In all, 33 animals were used in this investigation. Of these, 12 were used in the control series, 13 in the hypothermic series, and 8 in the hypothermic-operative series.

1. *The Control Series.*—In the control series, 12 dogs were operated upon, the result being recorded in Table 1. Four animals had their porta hepatis, celiac axis, and superior mesenteric artery occluded for 20 minutes; four, for 30 minutes, and four,

TABLE 1.—*Results of Control Hepatic Inflow Occlusion*

Time of Occlusion, Min.	No. of Dogs	No. of Recoveries	Per Cent of Recoveries
20	4	4	100
30	4	2	50
40	4	0	0

for 40 minutes. When these vessels were occluded, the whole of the small bowel and the proximal 3-4 in. (7.5-10 cm.) of the large bowel immediately became a white, cadaverous color. The liver slowly turned a light-brown and the spleen a dark-gray color.

All dogs in the 20-minute control group made uneventful recoveries. On release of the clamps, the only untoward finding was a mild edema of the small bowel and proximal colon. No abnormalities were noted at the time of killing, two weeks later.

Two of the dogs subjected to occlusion for 30 minutes recovered after a stormy postoperative course, requiring intravenous therapy for two days to combat severe vomiting and diarrhea. The other two animals died on the second postoperative day.

At operation, on release of the occluded vessels, the following observations were made on the 30-minute control series:

1. The peritoneal cavity contained from 40-60 cc. of serosanguineous fluid.

2. The liver slowly regained its normal color except in the fatal cases, in which the liver had a mottled appearance with numerous engorged areas.

3. Small hematomata were observed in the greater omentum and peritoneal reflections.

4. The cardiac end of the stomach contained many small hematomata in the area supplied by the left gastric artery in those animals in which it had been patent.

5. The small bowel became very edematous, as did the proximal 3-4 in. of the large bowel.

6. Peristalsis was observed in all cases.

7. In the two fatal cases many small, pinpoint hemorrhages were noted in the wall of the small bowel.

Autopsy on the two dogs which survived at the time they were killed, three weeks later, failed to reveal any abnormalities. In the fatal cases 100 cc. of serosanguineous fluid was found in the peritoneal cavity. The same hematomata which were noted at operation in the wall of the stomach, small bowel, and peritoneal reflections were present. The mottled appearance of the liver also remained unchanged from its condition at operation.

In the 40-minute control series all dogs died within 55 minutes of the release of the clamps. At operation the changes observed were much the same as those noted in the fatal cases in the 30-minute control series, except for the following changes:

1. The small bowel became extremely edematous and the hematomata in the bowel wall were much larger, being about 2 cm. in size.

2. No peristalsis was observed.

3. There were also areas of swelling in the small bowel that appeared to be subserosal areas of gas.

Autopsy findings were identical with operative findings, due to the short period of survival.

2. *The Hypothermic Series.*—In the hypothermic series, 13 dogs were subjected to occlusion of the circulation of the liver, the results being recorded in Table 2. One dog died at operation, owing to a technical error which was unrelated to the investigation. Of the remaining 12 animals, 2 were subjected to hypothermia only, in order to gain familiarity with the technique. Both dogs made uneventful recoveries and when they were killed, two weeks later, no abnormalities could be found.

Five dogs were subjected to occlusion of the vascular supply of the liver for 60

TABLE 2.—*Results of Hypothermic Hepatic Inflow Occlusion*

Time of Occlusion, Min.	No. of Dogs	No. of Recoveries	Per Cent of Recoveries
60	5	5	100
90	5	1	20

minutes under hypothermia at 28 C. All made uneventful recoveries. At operation the changes were very similar to those observed in the 20-minute control series, with mild edema of the small bowel and proximal 3-4 in. of the large bowel following release of the occluded vessels. Autopsy at the time the animals were killed, two to four weeks later, failed to demonstrate any abnormalities.

Occlusion of the circulation to the liver for 90 minutes was performed on a series of five dogs, with uncomplicated recovery in only one case. The other four all survived at least 15 hours following the release of the occluded vessels. Operative findings were almost identical with those observed in the 40-minute control series. The peritoneal fluid, the mottled appearance of the liver, the large hematomata of the small bowel and the peritoneal reflections, and the subserosal areas of gas in the bowel wall were all indistinguishable from the findings in the 40-minute control series.

One animal survived 21 days and was of great interest. It required intravenous therapy for three days to combat severe vomiting and bloody diarrhea, both of which were features of all the fatal cases in this series. After three days the vomiting and diarrhea ceased, but at no time did the dog regain its appetite or appear normally active. It continued to lose weight and after two weeks developed a cold that went on to pneumonia, which was not improved by antibiotic therapy. The animal died 21 days postoperatively. At autopsy there were obvious signs of weight loss. The peritoneal cavity contained 100 cc. of straw-colored fluid. The liver presented five large areas of soft, mushy necrotic material. The bowel wall was contracted and white in color except for three round hematomata, about 2 cm. in diameter.

The animal which survived 90-minute occlusion showed minimal changes at operation, and at autopsy, one month later, no abnormalities could be detected. It was of interest that this animal and the one that survived three weeks both came from a different animal house and were obviously younger and in far better nutritional condition than the other animals.

3. *The Hypothermic-Operative Series.*—In this series, dogs under hypothermia at 28 C, with the vascular supply to the liver occluded for 60 minutes, were subjected to cholecystectomy and resection of the right lobes of the liver. In one case splenectomy was also performed.

Eight animals were subjected to this operative procedure, one of which died at operation when too much traction was put on the liver and one of the hepatic veins was torn from the inferior vena. Of the remaining seven dogs, all made uneventful recoveries, as shown in Table 3.

TABLE 3.—*Results of Hypothermic and Operative Series*

Time of Occlusion, Min.	No. of Dogs	No. of Recoveries	Per Cent of Recoveries
60	8	7*	87.5

* One dog died at operation, owing to a technical error.

The minimal bleeding at operation was very striking. Active bleeding was absent once the hepatic veins were clamped. The only blood loss appeared to be the blood that was already in the liver prior to occluding the porta hepatis, celiac axis, and superior mesenteric artery. Once the initial blood was wiped away, the cut surface was surprisingly dry and remained so even after release of the occluded vessels.

At the time the animals were killed, four to six weeks later, no abnormality could be detected other than the fact that the left lobes of the liver had become greatly enlarged. A careful search was made in all cases to see whether the bulldog clamp had caused any damage to the common bile duct. In no case could any pathological changes be found.

4. *Blood Pressure Determinations.*—Blood pressure recordings were taken on all animals, and, except for minor variations, they were very similar.

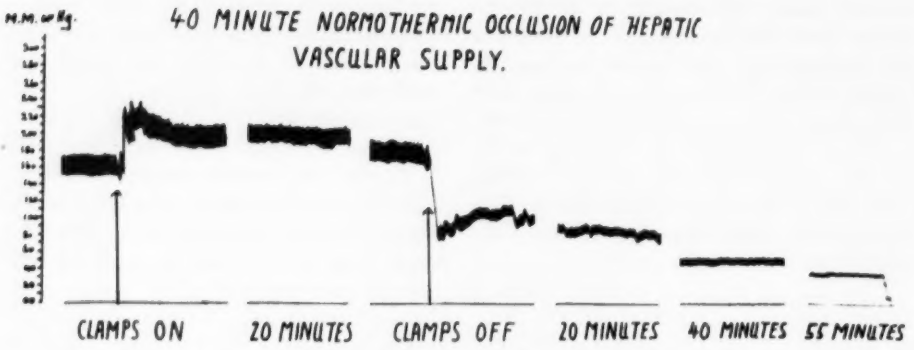

Fig. 1.—Blood pressure recording of Dog 11: 40-minute normothermic occlusion of hepatic vascular supply.

The control animals in which the porta hepatis, celiac axis, and superior mesenteric artery were occluded for 20 minutes demonstrated a very marked rise in blood pressure on occlusion of these vessels. A marked drop in pressure and rapid return to normal levels on release of the occluded vessels was a constant finding in this series.

The findings in the 30-minute control series were very similar to those of the 20-minute control series. The only difference was that following the fall in blood pressure on release of the occluded vessels the return to normal levels was slower.

In the 40-minute control series following the release of the occluded vessels the blood pressure showed a continuous slow downward course until the animal died (Fig. 1).

Figure 2 shows the recording made on a hypothermic animal in which the circulation to the liver was not occluded. This tracing demonstrates the characteristic increase in blood pressure during the early phase of hypothermia (35 C), followed by a decrease below the normal precooling level as the body temperature is lowered. Finally, the blood pressure becomes comparatively stabilized at a low level at 28 C. These findings are in agreement with those of Bigelow.

Figure 3 shows the effect on the blood pressure under hypothermia of 28 C when the porta hepatis, celiac axis, and superior mesenteric artery were occluded for 60 minutes. At 90 minutes of occlusion and 60 minutes of occlusion with cholecystectomy and resection of the right lobes of the liver, the recordings were quite similar. They all showed a fairly marked rise in blood pressure on occlusion of the circula-

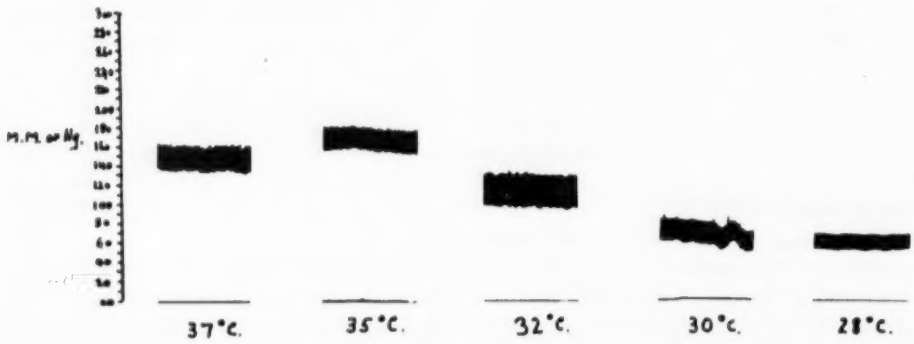

Fig. 2.—Blood pressure recording of Dog 13, with hypothermia only.

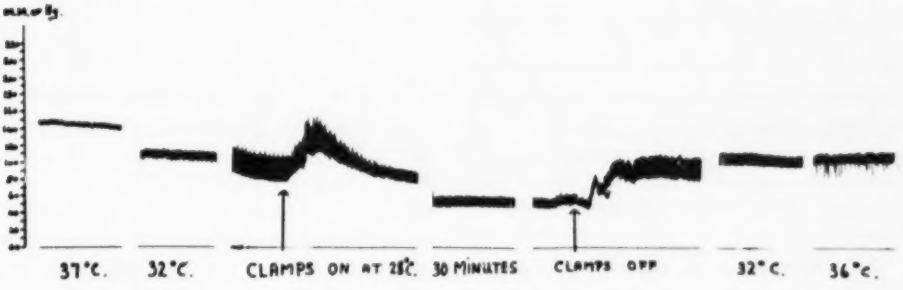

Fig. 3.—Blood pressure recording of Dog 17: 60-minute hypothermic occlusion of hepatic vascular supply.

tion to the liver, although not as marked an effect as was noted in the control series. The drop on release of the occluded vessels was also very slight and was rapidly followed by a rise in blood pressure.

Comment

A word should be said in explanation of the level of 28 C which was selected for the experimental hypothermia series. Unquestionably, a longer safe interval of occlusion could be obtained by working at lower body temperatures. However, below 28 C the complications of cardiac arrhythmias and of ventricular fibrillation enter the picture, and we were anxious to test a level which was relatively safe from this standpoint, while still hoping for a significant prolongation of the safe time of occlusion. The experience of operating in a bloodless abdominal field is quite unique, and such a field is not easy to visualize until one has actually experienced it. One hour of operating time is equivalent to several hours under ordinary conditions, not so much because of time saved in clamping and tying, but because of the speed with which dissection can proceed with safety. Accordingly, we feel that an apparently well-tolerated technique which allows of the interruption of the hepatic flow for 60 minutes is a worthwhile contribution.

With regard to the method of cooling employed, our combination of very limited surface cooling and intravenous lytic agents has not been widely used on this continent. Although the technique may smack of polypharmacy, we found that no component of the lytic cocktail could be omitted without the development of shivering or restlessness and delay in cooling, the only alternative being to use much larger doses of anesthesia, with resultant delay in recovery of consciousness.

The intravenous use of chlorpromazine and promethazine calls for care as to precise dosage. These drugs appear to be much more potent when given by the intravenous route, and doses which appear homeopathic have marked effects. Our original dose used in most of the experiments reported in Part I consisted of 50 mg. of each drug to the cocktail of 20 cc. We found later that exactly half that dose was quite adequate and that animals recovered consciousness much more promptly on the smaller dose.

A distinct impression was gained that our experimental animals displayed a marked resistance to surgical shock, as has been pointed out by Laborit.[12] This resistance was particularly striking in the total-evisceration experiments reported in Part I, but was also noted in this group. Further studies on this important effect are being undertaken in our laboratory.

The 20-minute period, which was found to be the maximum safe time of occlusion in normothermic dogs, is in general agreement with the opinion of others.[6] The 60-minute period safely achieved with clamps on the superior mesenteric artery, celiac axis, and porta hepatis, at 28 C, conforms

with the report of Bernhard and colleagues.[3] Our third group demonstrates good tolerance of the animals for extensive partial hepatectomy during the period of occlusion. It also indicates that the liver retains its integrity to a degree commensurate with normal convalescence of the animal and adequate regeneration of the nonresected remnant.

An observation of importance was the complete absence in any of our experiments of any unusual hemorrhagic tendency. Neither during the cooling period, after release of the clamps, nor subsequently was any troublesome bleeding encountered. Specific hematological studies were not undertaken. It is possible that the absence of this complication is related to the less traumatic method of cooling, as opposed to total immersion in ice water, which is commonly used.

The blood pressure tracings serve to indicate the relative stability of pressure in the experimental series as compared with the normothermic controls. It is a matter of conjecture at present as to how much of this stabilizing effect is due to the hypothermia itself and how much to the action of the lytic drugs.

The technique of liver resection which was developed and is described above consisted in simple dissection through dry liver parenchyma and suture ligature of the pedicle of each lobe. It proved simple, safe, and rapid, apparently free from the many difficulties outlined by Warvi in his review of methods of liver resection.[10] It goes without saying that the human liver and the liver of the dog are not completely comparable in their surgical anatomy. Nevertheless, we feel that the general method of vascular occlusion with general hypothermia may now be submitted to clinical trial.

Summary

The vascular inflow to the liver was occluded by clamping the celiac axis, the superior mesenteric artery, and the entire porta hepatis, including the portal vein, hepatic artery, and common bile duct. The maximum safe time of such occlusion in dogs at normal body temperature was found to be in the neighborhood of 20 minutes.

The procedure was repeated in an experimental series of dogs cooled to 28 C by means of limited surface cooling and a lytic cocktail of chlorpromazine (Largactil), promethazine (Phenergan), and meperdine (Demerol). By this means the maximum safe time of occlusion was raised to 60 minutes.

In a third series, during a 60-minute period of occlusion in hypothermic animals, an extensive partial hepatectomy of over 50% of the liver was performed, together with cholecystectomy. All animals tolerated the procedure without fatality and made rapid recoveries.

Conclusions

1. Occlusion of the celiac axis, superior mesenteric artery, and porta hepatis produces a bloodless liver satisfactory for major resectional surgery.

2. A full hour of occlusion of the vascular inflow to the liver is well tolerated by animals rendered hypothermic to the comparatively safe level of 28 C.

3. During a 60-minute period of occlusion, a resection of the right half of the liver and the gallbladder may be performed with ease. The animals recover rapidly, and the liver remnant appears to function satisfactorily, undergoing regeneration in the usual way.

4. The method of inducing hypothermia using limited surface cooling combined with thiopental (Pentothal) and a lytic cocktail appears to possess the following advantages: (*a*) freedom from shivering, (*b*) apparent resistance to surgical shock, (*c*) reduced swings of blood pressure on applying and releasing vascular clamps, and (*d*) freedom from hemorrhagic complications.

5. The technique described would appear ready for clinical trial, particularly in the

treatment of primary neoplasms of the liver and gallbladder.

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Necrotizing Properties of Metaraminol (Aramine) and Arterenol (Norepinephrine)

Comparative Study

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Two vasopressors currently used in the treatment of various acute hypotensive states are metaraminol (Aramine; *l*-1-[*m*-hydroxyphenyl]-2-amino-1-propanol) and levarterenol (Levophed; *l*-norepinephrine). The cardiovascular and renal hemodynamics of these two drugs have been extensively studied by many investigators, both clinically and in experimental animals.[1-3] Their local effects on the soft tissues, when administered intravenously or subcutaneously, have received inadequate attention, although in many patients the local necrotizing properties of various vasoconstrictors have been distressingly obvious.[4]

In a previous report,[5] it was shown that soft-tissue sloughs associated with the intravenous administration of solutions containing arterenol were apparently due to extravascular infiltration of the solution. In addition, it was demonstrated that "Levophed sloughs" could be prevented by injecting phentolamine (Regitine) into and about the margins of the extravasation. Moyer[6] and others[7,8] have not observed deleterious local effects from the infiltration of soft tissue by solutions containing metaraminol. Since both these drugs are being used on a large scale, further evaluation and comparison of the local toxic manifestations of metaraminol and levarterenol on the soft tissues seemed indicated.

Moyer and associates[6] found that on a weight basis metaraminol is only about 1/20 to 1/25 as potent a hypertensive agent as arterenol when administered by continuous intravenous infusion to patients in shock. Metaraminol has been found to have an epinephrine pressure ratio of 18:1 (by weight).[9] Grollman[10] noted that levarterenol is 1.64 times as active as a pressor compound as metaraminol.

Experimental Investigation

As it was impossible to perform controlled experiments of this type on humans, dogs were selected for study.

A separate test solution of each drug was prepared. The first solution consisted of 300 mg. of metaraminol bitartrate in 1000 cc. of 5% dextrose in water. The second solution consisted of 4 mg. of levarterenol bitartrate in 1000 cc. of 5% dextrose in water. In the following experiments the local effects on the soft tissues of these two test solutions were noted.

Intravenous Infusion.—In five dogs, 200 cc. of the levarterenol bitartrate solution was continuously infused into peripheral, medium-sized veins for a period of three hours (Table 1). Similarly, five dogs received 200 cc. of the metaraminol test solution (Table 2). The intravenous administration of each solution resulted in marked spasm of the veins receiving the infusion. Soft-tissue sloughs could not be produced by this method of administration. Previous studies[4,5] demonstrated that the levarterenol solution could be continuously infused intravenously for 72 hours without producing soft-tissue necrosis.

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From the Whitehead Department of Surgery, Emory University School of Medicine, and Grady Memorial Hospital.

TABLE 1.—*Effect of Levarterenol Bitartrate on Soft Tissues*

Drug	Method of Administration	Dosage	Number of Dogs	Number of Sloughs
Levarterenol	Intravenously	800 γ in 200 cc. 5% dextrose in water	5	0
5% dextrose in water	Subcutaneously	200 cc.	10	0
Levarterenol	Subcutaneously (rapidly)	8 mg. of concentrated levarterenol	2	0
Levarterenol	Subcutaneously (rapidly)	400 γ in 100 cc. 5% dextrose in water	12	0
Levarterenol	Subcutaneously (slowly)	400 γ in 100 cc. 5% dextrose in water	12	12

Single Subcutaneous Injection.—The local effects of the rapid subcutaneous injection of metaraminol and levarterenol solutions were studied in 5 and 12 dogs, respectively. Single, forceful injection of 100 cc. of levarterenol bitartrate solution into the subcutaneous tissue did not result in soft-tissue sloughs. The skin overlying the site of injection very rapidly became cold and extremely white, the changes gradually disappearing as the solution was absorbed. The local changes following the rapid injection of an equal quantity of metaraminol bitartrate (30 mg.) solution were similar, but not as pronounced as the changes produced by the levarterenol solution.

The effect of drug concentration was further evaluated by injecting 8 mg. of concentrated levarterenol (2 ampules of levarterenol (Lævophed) and 100 mg. of undiluted metaraminol bitartrate, respectively, into the subcutaneous tissues of two dogs. Slight swelling and blanching of the tissues occurred at the site of injection, but necrosis did not result. Thus the duration of the infusion, rather than the concentration, appeared to be the critical factor.

Slow Subcutaneous Infusion.—One hundred cubic centimeters of levarterenol bitartrate solution was given by drip infusion into the subcutaneous tissues of the medial thigh of 12 dogs. Whereas the rapid injection of 100 cc. of levarterenol test solution failed to produce soft-tissue necrosis, the same concentration and quantity of solution slowly but continuously infused into the subcutaneous tissues for a period of one hour routinely resulted in the formation of a massive soft-tissue slough. Similarly, 200 cc. of metaraminol bitartrate (60 mg.) solution was continuously infused for 120 minutes into the subcutaneous tissue of the right thigh of five dogs. As a control, an equal quantity of 5% dextrose in water was infused into the left thigh. Sloughing of tissue did not result. An additional five dogs were similarly studied except that 300 cc., instead of 200 cc., of the metaraminol solution was used. The local effects of massive extravasation of metaraminol solu-

TABLE 2.—*Effect of Metaraminol (Aramine) Bitartrate on Soft Tissues*

Drug	Method of Administration	Dosage	Number of Dogs	Number of Sloughs
Metaraminol	Intravenously	60 mg. in 200 cc. 5% dextrose in water	5	0
5% dextrose in water	Subcutaneously	200 cc.	10	0
Metaraminol	Subcutaneously (rapidly)	100 mg. of concentrated metaraminol bitartrate	2	0
Metaraminol	Subcutaneously (rapidly)	30 mg. in 100 cc. 5% dextrose in water	5	0
Metaraminol	Subcutaneously (slowly)	60 mg. in 200 cc. 5% dextrose in water	5	0
Metaraminol	Subcutaneously (slowly)	90 mg. in 300 cc. 5% dextrose in water	5	0

tion into the subcutaneous tissues were evanescent. Localized swelling of the regional tissues was produced, and the overlying skin became slightly cold and pale. As the fluid was absorbed, these signs gradually disappeared. In one dog, a mild rubor and ecchymosis of the skin developed at the site of the infusion, but these signs subsided during the succeeding three days. The local changes produced by metaraminol were in marked contrast to the dramatic temperature and color changes and to the large soft-tissue sloughs that subsequently formed when the levarterenol solution was slowly infused into subcutaneous tissues.

Summary

Metaraminol (Aramine) and arterenol (norepinephrine) are potent vasoconstrictors which are frequently used in the treatment of neurogenic hypotension. Soft-tissue sloughs resulting from the intravenous administration of solutions containing levarterenol are apparently due to, and can be experimentally produced by, the slow extravascular infiltration of the solution.

Within the limits of this study, soft-tissue necrosis could not be produced by infusing metaraminol intravenously or into the subcutaneous tissues. Metaraminol can apparently be given by intravenous drip infusion without the danger that inadvertent extravasation will lead to extensive soft-tissue sloughs.

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Severe Hypertension Due to Congenital Stenosis of Artery to Solitary Kidney

Correction by Splenorenal Arterial Anastomosis

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The following case of a child with severe hypertension due to stricture of the main artery to a solitary kidney is believed to be unique in that the hypertension was promptly relieved by end-to-side anastomosis of the splenic artery to the renal artery distal to the stricture and has remained continuously normal for the period of 14 months since operation.

Report of a Case

A white girl aged 10 years was admitted to the Ochsner Foundation Hospital on Nov. 20, 1955, because of headaches, dizziness, and epistaxis, associated with high blood pressure of two months' duration, which had been incompletely controlled by medical treatment. The initial blood pressure was 280 mm. Hg systolic and 180 mm. Hg diastolic. She had had no significant previous illness. A younger sister had been "blue" during the first two years of her life, but thereafter her color had been normal.

Routine examination of the blood and urine by one of us (R. B.) in September yielded normal results. No drop in blood pressure occurred during two phentolamine (Regitine) tests. Normal renal structures and function on the left were demonstrated in the intravenous pyelogram, but no function was apparent on the right. In the retrograde pyelogram an apparently normal ureter was demonstrable on the right, but there was no evidence of a renal pelvis. Early in October the right flank was explored. The right ureter was traced upward and found to terminate in a fibrous cord. The adrenal gland was normal. No kidney was present. Two weeks postoperatively peripheral right facial nerve paralysis developed. In spite of careful medical management, the hypertension persisted at 170 to 200 mm. Hg systolic and 110 to 160 mm. Hg diastolic (Fig. 1).

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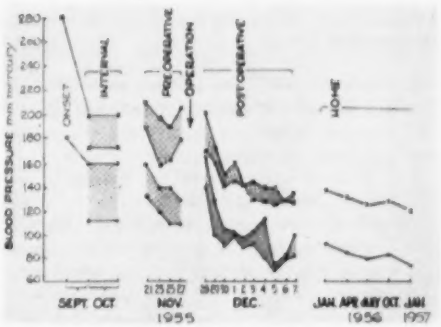


Fig. 1.—Graphic record of the systolic and diastolic blood pressures for a period of 3 months before and 14 months after left splenorenal arterial anastomosis distal to a stricture in the left renal artery. Stippled areas represent ranges of blood pressure.

The patient was sent to Ochsner Foundation Hospital for further study. On admission she appeared somewhat thin and pale. She was 50 in. (127 cm.) tall and weighed 67 lb. (30 kg.). The blood pressure was 200 mm. Hg systolic and 150 mm. Hg diastolic in all four extremities. On funduscopic examination mild spasm of the arterioles was noted, but there was no nicking, hemorrhage, exudate, or papilledema. Partial peripheral paralysis of the right seventh nerve was present. The lungs were clear. A moderately intense, blowing systolic murmur was heard to the left of the sternum, loudest in the third interspace.

Results of routine examination of the blood and urine were normal. An Addis count was normal. The fasting blood sugar was 60 mg/100 ml., and the blood urea nitrogen concentration was 14 mg/100 ml. The serum sodium was 141 mEq. per liter, and the serum potassium, 4.6 mEq. per liter. The tuberculin and Wassermann reactions were negative. No abnormalities were detected in roentgenograms of the chest and skull, and none in an electroencephalogram. Injection of 8.5 mg. of piperoxan hydrochloride resulted in rise of the blood pressure to 172 mm. Hg systolic and 120 mm.

Fig. 2.—In the intravenous urogram a normal pattern is seen in the left kidney but no excretion on the right side.

Hg diastolic, and then gradual return to the previous level of 154 mm. Hg systolic and 105 mm. Hg diastolic. Results of spinal fluid examination and pneumoencephalography were normal.

In the intravenous urogram, an apparently normal bladder and left ureter and a large, but otherwise normal, left kidney were noted (Fig. 2). The dye was promptly excreted on the left, but no dye was excreted on the right. In the translumbar aortogram made on Nov. 28, with use of a general anesthetic, the aorta appeared normal, but there was no evidence of a renal artery on the right side (Fig. 3). The left renal artery arose just distal to the celiac axis, and the proximal end was narrowed for a distance of 1.5 cm. Beyond this the artery enlarged to a diameter of 8 mm. It was shaped like a cornucopia and suggested poststenotic dilatation of the vessel. Branch arteries, particularly to the upper pole, appeared sparse and of small caliber.

As the child was already anesthetized, operation was performed through a left thoracolumbar incision. The adrenal gland was normal. The kidney was large but otherwise normal. The anatomic relations of the renal artery were the same as had been demonstrated radiographically. Close to the aorta the renal artery was extremely narrow. Distal to this point a thrill was present, and a cornucopia-like dilatation of the vessel was noted. The artery then divided into two main branches. The pressure in the vessel was palpably low. It was decided that end-to-side splenorenal arterial anastomosis would be the best way by which to by-pass the stricture (Fig. 4). The spleen was removed. The splenic artery was divided in its midportion. The proximal end was beveled, and five 00000 arterial nonabsorbable surgical (silk) sutures were placed in the end of the vessel and later sutured to the renal artery. An elliptical incision was made in the long axis of the clamped-off renal artery. The anastomosis was accomplished with the initial sutures and five additional interrupted sutures. The renal artery was occluded for 15 minutes. Removal of the clamps resulted in little oozing, and complete hemostasis was obtained with digital pressure. The orifice of the anastomosis measured 7 mm. in diameter, and excellent blood flow through it was apparent. The incisions in the diaphragm and parietes were closed. One drainage tube was placed in the left side of the thorax. During and immediately after operation the blood pressure rose to 230 mm. Hg systolic and 150 mm. Hg diastolic. The patient received 500 cc. of blood during the operation.

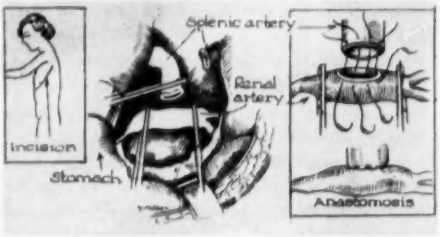
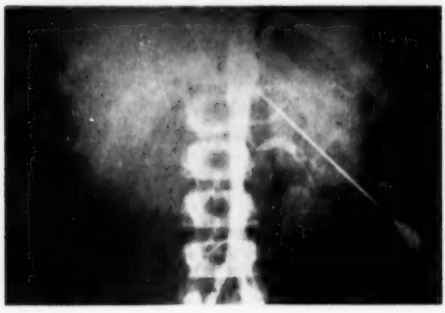


Fig. 4.—Technique of end-to-side splenorenal arterial anastomosis. Simple interrupted sutures of 00000 arterial silk are used.

The postoperative course was uncomplicated. The day of operation the patient passed 720 cc. of urine, and she never passed less than 700 cc. daily. Postoperatively, the urine contained no protein, sugar, red blood cells, or white blood cells. The specific gravity was 1.021. The blood pressure dropped rapidly (Fig. 1). It was significantly reduced by the second postoperative day (140 mm. Hg systolic and 88 mm. Hg diastolic) and remained at this level for four days. By the sixth day it had fallen to 128 mm. Hg systolic and 70 mm. Hg diastolic and remained in this vicinity for the rest of the hospitalization. Examination prior to discharge, on the 10th postoperative day, revealed no arteriolar spasm in the fundi, slight further im-

Fig. 3.—In the translumbar renal arteriogram absence of the right renal artery and juxta-aortic stenosis of the left renal artery with poststenotic dilatation of this vessel are demonstrated.



provement in the power of the right facial nerve, and a soft, blowing systolic murmur in the epigastrium. Shortly after discharge the blood urea nitrogen concentration was 10 mg/100 ml.

The child returned home to the care of one of us (R. B.). All symptoms had been relieved, and her blood pressure remained normal (Fig. 1). She returned to the hospital four months after operation for examination. Her general appearance was considerably improved. During this hospitalization the systolic blood pressure ranged from 114 to 140 mm. Hg and the diastolic from 80 to 116 mm. Hg, with an average of about 120 mm. Hg systolic and 98 mm. Hg diastolic. The soft, blowing systolic murmur in the epigastrium was no longer present. Translumbar aortography was attempted but was unsuccessful. The hemoglobin was 15.5 gm/100 ml., hematocrit 44% and blood urea nitrogen concentration 11 mg/100 ml. The values for CO_2-combining power, creatinine, calcium, phosphorus, and white cells in the blood were all normal.

The child has remained well since operation. Her blood pressure has remained normal (Fig. 1). Results of urinalysis have been repeatedly normal. In January, 1957, her blood pressure was 124 mm. Hg systolic and 82 mm. Hg diastolic in the right arm and 122 mm. Hg systolic and 70 mm. Hg diastolic in the left arm. She was 59 in. (150 cm.) tall and weighed 79.5 lb. (35.8 kg.). She had grown 8 in. (20 cm.) and had gained 12.5 lb. (5.6 kg.) in the 14 months since operation.

Comment

We have been able to find only two previous reports of cases of relief of hypertension due to renal arterial stenosis by conservative surgical procedures. In 1954 Freeman and co-workers [1] reported relief of severe hypertension in a man aged 46 for two years after endarterectomy, which corrected sclerotic stenosis of the left renal artery. This procedure was combined with endarterectomy of the aorta and iliac arteries. In November, 1955 (prior to the case here reported), Poutasse and colleagues [2,3] relieved severe hypertension in a boy 15 years old with bilateral stenosis of the renal arteries by insertion of arterial homografts between the aorta and the distal end of both divided renal arteries.

In 1952 Thompson and Smithwick [4] had unsuccessfully attempted to relieve hypertension associated with a sclerotic plaque occluding the left renal artery by end-to-end splenorenal arterial anastomosis. There was no function in this kidney either before or after operation. Normotension occurred after left nephrectomy. No statement was made concerning the patency of the arterial anastomosis at the time of the later nephrectomy. Hurwitt and co-workers [5] described prophylactic end-to-end anastomosis of the left splenic and renal arteries in a patient with recurrent aortic thrombosis extending up to the renal vessels; one year later slight rise in the patient's blood pressure was noted.

Our case is unique in that the hypertension was cured by correction of the stenosis in the artery to a solitary kidney. Occurrence of fusiform juxta-aortic stenosis in a child 10 years old is considered congenital. Nothing was found at operation to suggest inflammation or sclerotic degeneration.

The thoracoabdominal incision proved satisfactory. Dissections and operative experience since then have indicated that a transabdominal approach is feasible for operation on either renal artery. The splenectomy was, in retrospect, unnecessary, as only the proximal portion of the redundant splenic artery (Fig. 4) was used for the anastomosis. The splenic artery is commonly tortuous, and even loops on itself. It courses, at most, a few centimeters from the left renal artery. In these circumstances where a small-vessel shunt to a vital organ is required, particularly in a young person, we prefer an autogenous vessel to a homograft or a plastic graft. On the left side a splenorenal shunt is preferred. If a graft is required on the right side, one has the choice of swinging the divided splenic artery across, of using a transplanted segment of the splenic artery as a graft between the aorta and the renal artery, or of using a homograft or a plastic graft.

Whenever possible, we strongly recommend end-to-side anastomosis to the renal artery, because (1) the natural circulation through the renal artery is not destroyed, (2) disparity in the size of vessels or grafts

is immaterial, (3) if thrombosis or stenosis should develop at the site of anastomosis, the patient's condition is not worse than prior to operation, and (4) stenosis of the suture line in small vessels is less apt to occur with end-to-side anastomosis. Every effort must be made to prevent stenosis of the suture line, as this, obviously, would lead to recurrent hypertension. We prefer meticulously inserted simple end-on interrupted sutures of fine arterial silk.

Summary

A case is reported of severe symptomatic hypertension in a child 10 years old with congenital absence of the right kidney and stricture of the left renal artery successfully treated by end-to-side splenorenal arterial anastomosis distal to the stenotic portion. Normal blood pressure has persisted 14 months postoperatively.

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An Adjustable Aortic Graft

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Aortic replacement surgery has become a well-established procedure. Many substances have been used as vessel substitutes. The picture is slowly clearing; some substances have been tried and rejected; others continue to show promise.

Regardless of the type of graft available, the problem of proper size and shape remains. All too often an otherwise ideal procedure must be partially compromised by the necessity of adapting the graft available to the host's remaining vessels. Complicated plastic tailoring of the graft or host vessel becomes necessary to accomplish a functional anastomosis.

This is particularly true when homografts are used. The diseased aorta is often dilated and the diameter of the homograft, selected because of its lack of disease, is significantly smaller.

Our investigations have indicated that polyvinyl formalinized (Ivalon) sponge is superior to homografts for large-caliber vessels not traversing flexion creases. In this respect, we are in agreement with the recent excellent presentation by Mortensen and Grindlay,[1] in which they have recorded the properties of properly constructed Ivalon sponge tubes. Rob, in Great Britain, also has expressed his preference for Ivalon sponge as a substitute for large vessels.[3]

The illustrations indicate how a molded bifurcation graft can be adapted to a remaining proximal aortic segment of any diameter. This is achieved by a simple tapering of the aortic stem of the Y-graft. After the proper diameter is chosen, the aortic stem is cut at that level and the anastomosis performed. The iliac stems are then cut off at the proper level and anastomosed to the distal iliac segments of the host. The actual level of the bifurcation is insignificant, and it can be adjusted to the more important consideration of proper fitting of the aortic stem. If it is necessary to lengthen the prosthesis, we have shown experimentally that two polyvinyl formalinized sponge tubes can be anastomosed with satisfactory results.

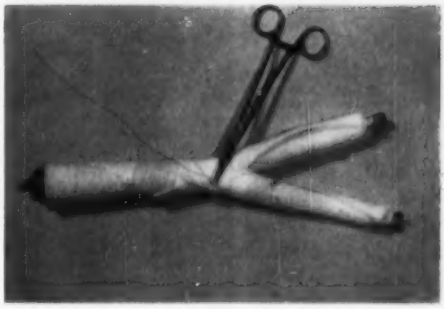
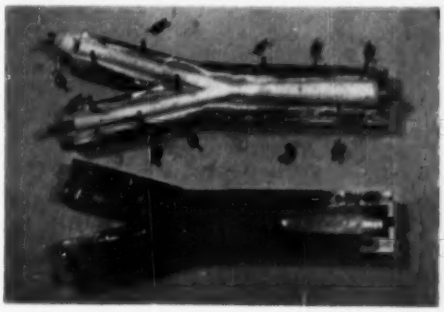


Fig. 1.—The moist sheets and strips of polyvinyl formalinized (Ivalon) sponge are rolled about the central brass core of the Y-mold.

Fig. 2.—The graft has been compressed and autoclaved, and is ready for removal from the mold.



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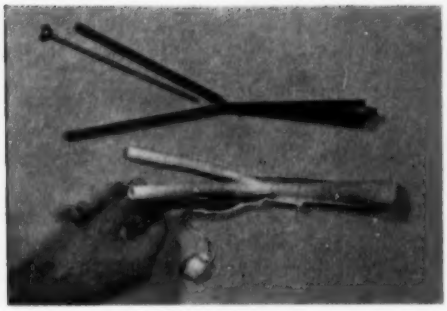

Fig. 3.—The graft is removed from the brass core and trimmed, and is ready for use.

Fig. 4.—The graft is in place; the superior anastomosis is a perfect fit.

The method of manufacture of a bifurcation graft is similar to the original description as recorded by Shumway et al.[2] The precision ground stainless-steel dies surround a brass core with a removable leg. This forms a graft with walls 2 mm. thick. At this time, we believe the optimum compression of the original sponge should be sevenfold. This is achieved by wrapping sheets and strips of wet sponge about the brass core, compressing the substance within the mold, and autoclaving for "fixation." The sevenfold compression results from applying four or five layers of polyvinyl formalinized sponge, which has been cut to a thickness of 2 to 3 mm. while in a dry state. It is more important to pay attention to the details of the limbs, rather than the crotch, as it is these portions which will participate in the anastomoses. If they are too thick, suturing is difficult and host tissue agglutination is lacking; if too thin, excessive oozing results. Excessive density in the crotch region is unimportant if the three peripheral limbs become well infiltrated and attached to the host vessels.

We have placed a polyvinyl formalinized sponge prosthesis in the abdominal aorta in six clinical cases. The first patient died of postoperative hemorrhage, presumably due to a defective graft, which was much less dense than the recommended sevenfold compression. One of the remaining five patients with satisfactory grafts died seven days postoperatively of causes unrelated to the graft. At autopsy the graft was intact and functioning. One patient died of a massive hemorrhage into the intestine caused by the erosion of a duodenal ulcer into the graft. This graft had been in situ for six months. The remaining three patients are in the 11th, 10th, and 7th postoperative months, respectively, and are clinically doing well.

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Ulceration of the Rectum and Terminal Portion of the Colon

Significance in Absence of Malignant Disease and Chronic Ulcerative Colitis

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The physician who makes many proctosigmoidoscopic examinations often encounters some form of ulceration of the rectum or terminal portion of the colon. In my practice the commonest causes of serious ulceration are malignant disease and chronic ulcerative colitis. Numerous other conditions, however, may result in the formation of ulcers or in a diffuse ulcerative process in the region. Many of these conditions are uncommon, some extremely so. Also, except from the standpoint of differential diagnosis, some of them are not significant.

The Search for the Cause

In the attempt to ascertain the causes of ulcerative processes of the rectum or sigmoid portion of the colon, thorough taking of the history is of primary importance. Often the patient will neglect to mention important facts, either because he has forgotten them or because he is not aware of their significance. Although the history of the present illness is always important, it may be a detail of the patient's past, sometimes of hidden importance, which will lead to correct diagnosis. At other times diagnosis will be aided chiefly by information obtained from general physical examination.

Ulceration of the rectum or terminal portion of the colon may be a primary disease. More frequently, however, it is secondary to another pathologic process. Principal among still other causes is trauma, which sometimes is the result of treatment.

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Ulceration as the Primary Disease

I agree with the view that the condition known as chronic ulcerative colitis usually begins as ulcerative proctitis and progresses orad in the bowel. In numerous cases, while following a patient's course for months or years, I have observed such progression. Less frequently, but fairly commonly, the ulcerative process remains confined to the rectum; then, since the colon is not involved in the condition, to call it ulcerative colitis seems unwarranted. Whether the condition becomes ulcerative colitis or remains ulcerative proctitis, however, the proctoscopic picture is the same except for extent of involvement. Typically, in both conditions the mucosa appears moth-eaten and diffusely granular, and it bleeds on slight trauma. Furthermore, in both occur remissions and exacerbations; in both the same anorectal complications (particularly rectal strictures and fistulous processes) may develop, and of both the cause is unknown. Consequently, it would seem reasonable to assume that the type of ulcerative proctitis under consideration here is an aborted form of the more extensive disease known as chronic ulcerative colitis. However, until the cause is known, the assumption will not be justified. One difference between the two conditions, it seems to me, is that under proper management ulcerative proctitis is more likely to subside completely. Even after years, however, it may recur.

Ulcerative proctitis of the type just described often will involve only the lower third to half of the rectum. Usually, the entire circumference of the rectal segment is concerned in the process, but occasionally it may be confined to a more limited area, such as one wall. Rarely, the ulcerative process may be confined to a segment of the sigmoid portion of the colon, in which case it is referred to as "ulcerative sigmoiditis."

The diagnostician must not be misled by the diffusely inflamed appearance of the mucosa that may attend simple diarrhea or may follow the taking of a harsh laxative or administration of a soapsuds enema. If he is in doubt, he would do well to make reexamination a few days after diarrhea has subsided, or at a time when a laxative or an enema has not been given.

Ulceration Secondary to Other Disease

When ulceration of the rectum or terminal part of the bowel is secondary to another condition, the other condition may be manifested also in the lower part of the bowel, elsewhere in the gastrointestinal tract, or outside the gastrointestinal tract.

Primary Disease Localized in Lower Part of Large Bowel

Ulceration of the lower portion of the rhoids, prolapse of the rectal mucosa, or rectum may accompany internal hemorrectal procidentia. Usually ulceration is attendant on protrusion of tissue through the anus, and it may vary from superficial erosion of the mucous membrane resulting from mechanical irritation to ulceration of part or all of the protruding mass from interference with its blood supply. When tissue is protuberant, difficulty in determining the nature of the process is unlikely. However, if a patient is seen when the tissues have receded to their normal positions, dependence must be placed on the patient's history to establish the cause of the ulceration.

Rectal ulcers also may be the result of abscesses and fistulas, which, in turn, originated in anal crypts. Much of the mucosa that overlies a submucosal abscess may undergo dissolution, and an acute ulcer may result. A chronic ulcer, usually small and often associated with a tuft of granulation tissue, may represent the secondary opening of an internal fistula. Since the ulcers mentioned, and the conditions that underlie them, frequently are unproductive of symptoms, the history often is not of diagnostic aid. However, careful palpation and inspection of the anorectal region usually will establish a connection with a crypt.

Primary Disease Not Localized in Lower Part of Large Bowel

Ulceration of the rectum, or the terminal portion of the colon, or both, may accompany a heterogeneous group of diseases localized elsewhere than in the lower part of the bowel. These diseases may be primary in the gastrointestinal tract or outside it.

Infections (including the so-called infestations).—Infectious diseases that have at least the potentiality of causing ulceration of the lower part of the intestine are bacillary dysentery, amebiasis, tuberculosis, lymphogranuloma venereum, syphilis, gonorrhea, schistosomiasis, balantidial colitis, and histoplasmosis. Nothing diagnostically characteristic can be seen on proctosigmoidoscopy alone; diagnosis depends either on isolation of the causative organism or on some specific test or other determination.

Bacillary Dysentery.—In the acute stage of bacillary dysentery, proctosigmoidoscopic findings are fairly characteristic. Of course, severity of these findings may vary, but, in the typical case, all of the mucous membrane examined is diffusely hyperemic, edematous, and the site of superficial, flat, sharply defined ulcers of various sizes and shapes. Between the ulcers the mucosa may be covered by a grayish membrane, composed of fibrinous exudate. If the disease lasts more than a month, it is considered chronic,[1] and by the end of this time much,

or all, of the diffuse mucosal inflammation may have subsided and only ulcers and scars may remain.[2] In the chronic form of the disease remissions and exacerbations may occur. During the first week of the disorder the causative bacilli (various species of Shigella) may be cultured from the stool. After that, however, it may be difficult or impossible to find these organisms even if the material for culture is removed directly from an ulcer. Felsen and Wolarsky,[1] in particular, expressed the belief that bacillary dysentery often goes unrecognized. They contended, also, that chronic ulcerative colitis may be a sequel of this disease, but the opinion is not generally held.

Amebic Colitis.—At the Mayo Clinic, in about a third of the cases of amebiasis, ulcers have been seen in the rectum, or lower part of the sigmoid, or both, on sigmoidoscopic examination. The ulcers caused by Entamoeba histolytica usually have a typical appearance, but this does not mean that they cannot simulate ulcers arising from other causes. Nor does it mean that the picture on sigmoidoscopy, in cases of amebic colitis, is always the same. The so-called typical amebic ulcer is round or oval, usually only a few millimeters in diameter, has sharply defined, elevated margins, and lies in a field of mucous membrane that appears normal otherwise. Many, or only a few, of these ulcers may be scattered throughout the rectum and lower part of the sigmoid. Although the mucosa between the ulcers usually is of normal appearance, occasionally it may appear to be inflamed in a patchy manner, possibly from some other cause. In acute cases, also, there may be excessive mucosal destruction, and the ulcers may take on irregular, "atypical" forms. To isolate the E. histolytica when ulcers are present usually is easy; material for microscopic examination can be removed, with biopsy forceps, from the base or margin of the ulcer.

Healing of amebic ulcers takes place extraordinarily rapidly following proper treatment. Unless ulceration has been extensive, no trace of the ulcers will be found on sigmoidoscopy a week or so after a course of treatment has been completed.

Ulcerative Tuberculosis.—Ulcerative tuberculosis of the terminal portion of the colon and rectum probably never has been a common condition, and now that new medications have proved beneficial in treatment of tuberculosis in general, it is extremely rare. When present, usually it is a complication of pulmonary tuberculosis, and, almost always, it is associated with tuberculous disease higher in the colon or in the small intestine. Also, it usually justifies an extremely poor prognosis. Usually a tuberculous ulcer lies in a field of mucous membrane which otherwise looks grossly normal, and, in this respect, it is like an amebic ulcer; however, tuberculous ulcers are larger, and their margins are flatter and more irregular. They do not necessarily follow an annular direction in the rectal wall, as tuberculous ulcers of the small intestine usually do.

Lymphogranuloma Venereum.—In cases of lymphogranuloma venereum, proctitis and ulceration are secondary to involvement of the rectal lymphatic structures and are associated with rectal stricture. The presence of other anorectal manifestations of this disease, including abscesses, sinuses, and fistulas, aids in diagnosis, but final diagnosis is made by the Frei test.

Syphilis and Gonorrhea.—To my knowledge, we of the Section of Proctology at the Mayo Clinic never have seen rectal ulceration as a result of syphilis. Also, although gonorrhea may cause proctitis, occasionally accompanied by superficial ulceration, probably we shall see it rarely now that treatment of gonorrhea with antibiotics is highly successful.

Schistosomiasis (bilharziasis).—Schistosomiasis, another infectious disease which may cause ulceration in the terminal portion of the bowel, is not endemic in this country. One reason that it is not, apparently, is that snails of the United States are not suitable intermediate hosts for development of the

causative organism (the Schistosoma, or blood fluke, of which three types are important: S. mansoni, S. japonicum, and S. haematobium). However, in this era of global warfare and travel, it is possible that a patient with this disease may be seen in this country.

My information concerning the proctoscopic findings in schistosomiasis is derived chiefly from an article of Recio.[3] Before ulceration takes place, multiple small areas of hyperemia, or mucosal ecchymosis, give the rectal mucosa an appearance of "measles." Soon tiny abscesses appear, and when these rupture, to expel eggs into the rectum, tiny ulcers are formed. These ulcers frequently are no more than a millimeter in diameter, have irregular margins, and stand out prominently in an area of mucosa that looks relatively normal. The ulcers may coalesce to form "wider areas of superficial erosion." Obviously, there is nothing pathognomonic about the appearance of the ulcers, and diagnosis can be made only by finding the eggs in the feces or in specimens removed for biopsy.

Balantidial Colitis.—As far as I have been able to determine, balantidial colitis has not been seen at the Mayo Clinic since Buie's experience with it, reported in 1937. Buie[2] stated that in each of his three cases discrete ulcers were "scattered about through the rectal and sigmoidal walls" and were found in fields of mucous membrane that appeared to be relatively normal. Except that the ulcers were large (they averaged about 1 cm. in diameter), they resembled, in all respects, amebic ulcers. Balantidial colitis is caused by the Balantidium coli, a large, ciliated protozoan, which commonly is found in the intestine of the pig, but rarely in that of man. When found in man, furthermore, it does not necessarily cause a pathologic condition.

Histoplasmosis.—Histoplasmosis, which is caused by infection with the fungus Histoplasma capsulatum, may be either a local or a general disease. The organ most frequently affected is the lung, but apparently the disease may involve any organ or combination of organs. In one case of generalized histoplasmosis which I have observed, there were a few scattered, discrete, shallow ulcers of the rectal mucosa. The diagnosis of histoplasmosis was made by biopsy of one of the ulcers. Weiss and Haskell[4] have reported a case in which the rectal lesions were polypoid rather than ulcerous.

Other Infections.—Ulceration of the terminal part of the bowel occasionally may be caused by infections that have not been mentioned here.

Noninfectious Diseases.—Leukemia and agranulocytosis may cause ulceration of the rectum, usually in combination with ulceration of the anus. My experience has been that anorectal ulceration associated with these diseases usually is of grave prognostic significance. Endometriosis involving the rectovaginal septum or sigmoid occasionally may be associated with superficial ulceration of the overlying rectal or sigmoidal mucosa. It also has been reported, on the basis of findings at necropsy,[5] that ulceration of the gastrointestinal tract, including the rectum, may accompany uremia. However, I have not seen a report of uremia and ulceration being found associated in a living person.

Regional Ileitis.—The finding of ulcers in the portion of bowel that is within reach of the sigmoidoscope often constitutes a clue to diagnosis of regional ileitis. I have come to think of these ulcers as being of three types, all of which can be found in a single patient. Ulcers of one type are large and indolent, are found in the anorectal region, and may occur spontaneously; they also frequently are the result of anorectal surgical measures. Ulcers of another type are large, irregular in shape, and usually shallow, and commonly only one or two are found in a single case. This type of ulcer is likely to be found low in the sigmoid portion of the colon. Ulcers of the third type are usually numerous and often are scattered throughout the rectum and that portion of sigmoid that is seen on sigmoidos-

copy. These ulcers are small, discrete, appear punched-out, and are indistinguishable, on gross inspection, from those typical of amebic colitis. Because more of my patients have regional ileitis than have amebic colitis, when I see ulceration of this third type I suspect ileitis first and amebiasis second. When examinations of stools and biopsy of material from the ulcers do not reveal parasites, treatment with amebacides causes no change in appearance of the ulcers.

Ulceration Related to Examination, to Treatment, or to Content of Large Bowel

Acute or chronic ulceration of the terminal portion of the bowel may be the result of previous examination or local or general therapeutic measures. Recognition of such ulcerative processes usually is aided chiefly by the history, but the situation and appearance of the processes frequently are characteristic. Also of diagnostic significance is the fact that, unless damage has been extensive, most of the lesions heal promptly, spontaneously, and without complication.

Causative Local Measures

Ulceration due to local measures may result from trauma incident to enemas, to previous instrumentation, or to treatment instituted for conditions of the rectum, sigmoid, or neighboring organs.

Enemas.—Most patients, as well as many persons who administer enemas, are ignorant of the acute angulation at the anorectal junction and of the fact that injury to the rectum may result in little or no pain. Therefore, the rectal wall may be traumatized by inserting the enema applicator too far or too forcibly. Usually the rent penetrates only the mucosa, is small, and is situated on the anterior wall just above the dentate margin. Occasionally, a linear mucosal tear may extend from a point just above the anus to the upper portion of the rectum. Rarely, the applicator may perforate the entire rectal wall.

When that occurs, a considerable amount of the solution may be forced into the extrarectal tissues. Thereupon, large extrarectal spaces are formed and extensive sloughing and ulceration of the rectal wall may take place. The patient may not be aware of the accident when it occurs. However, questioning may elicit the information that administration of the enema was attended by considerable discomfort and was followed by passage of blood from the rectum. Although recovery may be complete, disability is of long duration.

Ulceration of the rectal and sigmoidal mucosa also may be caused by enema solutions which are too strong chemically and, of course, by solutions which are hot enough to produce a burn. Fortunately, examples of either of these types of trauma are rare. Severe proctosigmoiditis may follow use of enemas containing a high concentration of hydrogen peroxide for treatment of fecal impaction. Early after such injury the clinical course and sigmoidoscopic findings simulate those of severe chronic ulcerative colitis. However, recovery can be expected, and the only residual pathologic finding probably will be mild to moderate generalized contracture of the rectum and sigmoid.

Previous Instrumentation.—In the course of sigmoidoscopy, bits of mucosa may be scraped from the wall of the bowel. This is most likely to occur at points where the bowel is angulated, particularly at the level of the rectosigmoid, where the bowel is in close proximity to the sacral promontory. Such trauma usually is significant only from the standpoint of diagnosis, when the mucosal defect is observed on subsequent examination.

Treatment of Rectal and Sigmoidal Lesions.—Ulceration may result from several kinds of treatment of rectal or sigmoidal lesions, and, again, the ulceration frequently is of diagnostic significance only. It may follow injection treatment for hemorrhoids or rectal prolapse. It may be due to

an incompletely healed surgical wound. Eradication of a neoplasm by fulguration or radium always is followed by temporary ulceration. Of special significance is the failure of a surgical wound of the rectum to heal; as previously intimated, this may be the result of inflammatory disease of the bowel (terminal ileitis or segmental colitis) beyond the reach of the sigmoidoscope.

Treatment of Lesions of Neighboring Organs.—"Factitial proctitis" is the term used to designate a condition of the rectal wall that may occur following irradiation for lesions involving neighboring organs. Usually it is the aftermath of application of radium to a malignant growth of the uterine cervix, and, in most cases, it is limited to the anterior wall of the rectum. The pathologic change in factitial proctitis varies from simple proctitis to true ulceration. Sometimes the rectovaginal septum is perforated. A significant finding in any case is the presence of telangiectasia of the involved rectal mucosa, particularly in the later stages of the process. These telangiectatic vessels remain a possible source of bleeding even after healing of the primary lesion.

Colostomy with Inactivity of Bowel Caudad to Site of Operation.—I have long been impressed by a condition of the mucous membrane which develops from prolonged inactivity of the portion of the bowel distal to a colonic stoma. Frequently the mucosa becomes reddened, often appears granular, and bleeds from slight trauma. In other words, its appearance resembles that which is characteristic of mild, chronic ulcerative colitis. I have no explanation for this change in the mucosa following colostomy. Although the condition is not true ulceration, I believe it should be included in this discussion.

Ulceration Caused by Contents of the Bowel or Removal of the Contents.—Ulceration of the terminal part of the bowel may be caused by impacted feces or foreign bodies. The trauma may result either from the presence of these agents themselves or from trauma incident to their removal. Obviously, there is usually no problem in diagnosis if the patient is seen before removal or escape of the impacted material or foreign body. If a patient is seen later, however, the cause of the ulceration can be ascertained only from the history.

Causative General Treatment

Evidence accumulated in recent years points to certain systemic medications as possibly causative of ulceration in the gastrointestinal tract.

Cortisone and Related Steroids.—Cortisone and cortisone-like steroids have been implicated by several authors as causative of gastrointestinal ulceration. In 1953, Sauer, Dearing, and Wollaeger[6] reported from the Mayo Clinic that in 10 cases "serious untoward gastrointestinal manifestations possibly related to administration of cortisone and corticotropin" had taken place and that in 3 of these cases ulceration was seen on sigmoidoscopic examination. In two of the three cases the ulcers were large and appeared in an otherwise normal mucosa; in the third, the pathologic condition was manifested by diffuse, granular proctitis of the lower third of the rectum. The sigmoidoscopic examinations in these cases were made by members of the Section of Proctology, of which I am a member, and, as far as I know, we have seen no other similar cases. In commenting on the findings, Sauer, Dearing, and Wollaeger[6] stated:

> Although it is possible that the occurrence of gastrointestinal complications in patients receiving cortisone or corticotropin is entirely by chance, the evidence from our study and from reports in the literature strongly suggests that administration of these hormones may be a causative factor. That it is not the only factor is apparent from the large number of patients who receive these agents and who do not experience any such complications.

Antibiotics.—Other implicated medications are the so-called broad-spectrum antibiotics. It is well known that use of these substances, either orally or parenterally, may result in diarrhea. Probably the diarrhea

is most frequently the result of chemical irritation of the intestine, but it may also be the result of infection by micrococci (staphylococci) [7] or Monilia, both microorganisms that have become prominent in the changed intestinal flora. Turell and Maynard [8] have reported on three cases in which sigmoidoscopic examination disclosed a "reddened, injected, edematous mucosa with tiny, scattered superficial ulcerations." Gram stains of smears taken from the mucosa in these cases revealed micrococci. Two of the patients had been given chlortetracycline hydrochloride (Aureomycin hydrochloride); the other had received oxytetracycline hydrochloride (Terramycin hydrochloride). There is also evidence that pseudomembranous enterocolitis may in some instances be caused by an upset in the intestinal flora caused by use of antibiotics. However, considering the wide usage of these drugs, such untoward reactions are rare.

Summary Not Summarized

In this paper I have attempted to group, in orderly fashion, diverse ulcerations of the rectum and terminal portion of the colon, exclusive of those that accompany malignant disease and chronic ulcerative colitis. I do not pretend that the consideration is exhaustive. Since the whole paper is a summary of a vast subject, I shall not attempt a summary of the summary.

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Investigations on Adsorption of Benzalkonium Chloride U. S. P. by Skin, Gloves, and Sponges

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Introduction

The wide clinical use of benzalkonium chloride U. S. P. as a germicide makes the investigation of its adsorption on articles immersed in it of practical, as well as theoretical, significance. Sporadic instances of isolation of bacteria from solutions supposedly containing benzalkonium have occurred. Subsequent tests on isolated organisms have shown them to be sensitive to the quaternary, indicating that their survival may have been due to a depletion of the germicide from the original solution.

A number of investigators have described the adsorption of quaternaries upon textiles. Goldsmith et al.[1] reported that chlorine and benzalkonium chloride are removed from solution by wool and cotton fabrics to a degree, depending on original concentrations. Fischer and Seidenberg[2] noted a definite correlation between antibacterial activity of water-soluble substances and their affinity for wool, a greater affinity representing a higher bactericidal action. Two quaternary compounds, Sapamine KW Concentrate (quaternary ammonium salt of diethylaminoethylamide hydroacetate, Ciba) and Desogen (dimethyl-[2-*N*-methyldodecanamidoethyl] [phenylcarbamoylmethyl] ammonium chloride, Geigy) were among those investigated. Latlief et al.,[3] primarily concerned with residual bacteriostatic action of quaternary-treated diapers, found that time and temperature of exposure, as well as concentrations of the quaternary in solution, were important factors in securing a sufficiently high adsorption to inhibit ammonia production from urea by Proteus mirabilis for definite periods. Since 1937, the substantivity of quaternary ammonium salts has also been appreciated in the textile industry, where processing for water repellence and antistatic properties includes their use.[4]

The purpose of this study was to determine how much quaternary is adsorbed by articles such as gauze sponges, cotton balls, and rubber gloves. This information would provide a guide to the preparation of solutions of sufficient concentration to ensure a bactericidal residual in the liquid in which these articles are immersed.

The amount of quaternary removed from solution by skin of the hands and of the back was also determined. The practical implications of this information on preoperative skin disinfection are obvious.

Method

The two-phase titration method, as described by Lewis and Herndon,[5] was used to determine the amount of quaternary in solution before and after immersion of articles. The advantages of the two-phase method are rapidity and simplicity; the only equipment necessary is a buret and volumetric pipettes. A solution of methylene blue is added to the benzalkonium chloride solution; chloroform is then introduced, with the resultant formation of two layers. Upon titration with a standardized dodecyl (lauryl) sodium sulfate solution, the excess anionic detergent forms a chloroform-soluble dye complex. At the end-point the colors in the two layers are the same. Lewis and Herndon[5] attribute a precision to this procedure of 2 parts per million variation from the mean over a range of 4 to 1000 parts per million.

Various materials were immersed in benzalkonium chloride solution for different periods of time. Since the volume of solution was known,

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the amount of compound taken out of solution could be calculated readily.

The substantivity of quaternary on the following materials was measured:

1. Zobec sponges, 2 by 2 in. (Johnson & Johnson)
2. Gauze sponges, 8 by 4 in., 12-ply (Johnson & Johnson)
3. Brown milled smooth rubber gloves, size 7½ (The Seamless Rubber Company, New Haven, Conn.)
4. Cotton balls, plain
5. Cotton balls, gauze-covered
6. Viscose rayon balls (Johnson & Johnson)
7. Hands
8. Skin of the human back

Measurement of the adsorption of quaternary by human skin was accomplished by layering known volumes of solution over an isolated area of skin. The isolation was effected by a vacuum seal about the periphery of a hole in a metal template.

Results and Comment

Adsorption is a phenomenon which depends on the attractive forces between atoms on the surface of the adsorbent (in this case, textile, rubber, skin) and the atoms of the material adsorbed, i. e., the positively charged cation of the quaternary ammonium salt. How this phenomenon is affected by various surfaces, and what influence temperature, concentration, and duration of contact have, are indicated in the accompanying Tables.

The amount of benzalkonium chloride adsorbed onto articles commonly immersed in such solutions is indicated in Table 1. The large sponge adsorbed almost six times the amount adsorbed by the small sponge; however, on a per gram basis, the adsorption is comparable: 3.2 and 3.7 mg. per gram, respectively. Viscose rayon balls showed an extraordinarily high substantivity on a per gram basis as compared with cotton. Goudie [6] states that the substantivity of viscose rayon fiber is 18.1 to 20.0 mg. of quaternary per gram from solutions ranging from 1:1500 to 1:500.

The figure for brown milled smooth rubber gloves was 41 mg. per glove. Simple arithmetic reveals that 65 sponges (8 by 4 in.), 12 pairs of rubber gloves, or 56 viscose rayon balls in a liter of 1:1000 solution can conceivably deplete the solution of quaternary. In actual practice, however, it is not feasible to place so many objects into 1 liter of solution; furthermore, the concentration of quaternary decreases with each addition, resulting in subsequently lower uptake. Nevertheless, substantivity of quaternary must be taken into consideration when making up solutions, so that a sufficiently high residual may be present in the immersing liquid for germicidal effect. Excess volume alone is insufficient protection where solutions are reused.

In order to approximate conditions of use, 100 Zobec sponges (2 by 2 in.) were placed into 750 ml. of an 0.134% solution (1:746) of benzalkonium chloride. After immersion for 72 hours, the concentration of the quaternary was titrated to be 0.094% (1:1064), a more than adequate residual.

The bactericidal action of a 1:750 solution in contact with cellulose was determined by inoculating each of six Zobec sponges

TABLE 1.—*Adsorption of Quaternary (Benzalkonium) from Solution on Immersed Objects* *

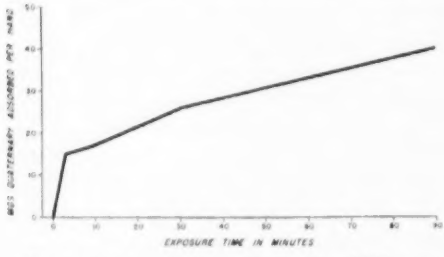
Object	Benzalkonium Chloride Concentration, Mg/Ml.		Benzalkonium Adsorbed, Mg.	
	Before Immersion	After Immersion	Per Object	Per Gram
Zobec sponges (50)	1.28	1.13	2.6	3.7
Sponges 8×4 in. (10)	1.28	1.10	15.3	3.2
Rubber gloves, brown (3 pairs)	1.26	0.97	41.1	....
Cotton balls, uncovered (15)	1.27	1.16	6.2	6.8
Cotton balls, covered (15)	1.27	1.10	9.6	3.9
Viscose rayon balls (10 large)	1.00	0.79	17.9	17.6

* Table 1 shows the uptake of quaternary in a 36- to 48-hour period of exposure at room temperature. The concentrations before and after immersion are indicated. In all cases, the volume was 850 ml. of solution for the number of objects indicated in parentheses.

TABLE 2.—*Quaternary Adsorbed by Hands Immersed in a Solution* *

	Time of Exposure, Min.	Concentration of Benzalkonium, Mg/Ml.		Benzalkonium Adsorbed per Hand, Mg.
		Original	Final	
Subject 1	3	1.030	0.995	14.88
	10	0.960	0.920	17.00
	30	0.940	0.880	25.50
	90	0.955	0.860	40.38
Subject 2	3	1.272	1.238	14.45
		1.280	1.245	14.88
	30	1.285	1.213	30.60
	90	1.274	1.179	40.38
Subject 3	3	1.280	1.238	17.85
		1.279	1.225	22.95
	30	1.276	1.178	41.65

* Both hands were immersed up to the wrist in 850 ml. of benzalkonium chloride solution. Hands were previously dipped into 95% ethanol to defat the skin surface and also to present uniformly treated skin for immersion.



Graph 1 (drawn from Table 2).—Milligrams of quaternary adsorbed as a function of time (Subject 1).

with 1 ml. of 3×10^8 suspension of Escherichia coli 198. All six sponges were then immersed in 100 ml. of 1:750 solution; the bacteria were destroyed within five minutes. This inoculum represents an extreme case of contamination, one which would not ordinarily be encountered.

As a result of these investigations, quaternary ammonium salt solutions in common use at the Peter Bent Brigham Hospital were modified to 1:750, rather than 1:1000, to allow a margin of safety.

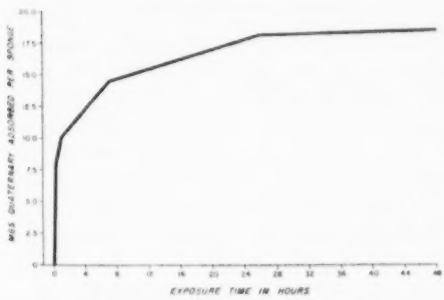
A considerable quantity of quaternary is absorbed by hands, as shown in Table 2. This process is a function of time. Within three minutes 36.8% of the amount taken up in 90 minutes is adsorbed.

Tables 3 and 4 indicate the results obtained with gauze sponges and viscose rayon balls over a period of 48 hours at room temperature. One set of each immersed in a refrigerated solution (4 C) and kept at that temperature for 48 hours showed a considerable difference from that at room temperature, a difference of 5.78 mg. per viscose rayon ball and 3.15 mg. per gauze sponge.

TABLE 3.—*Rate of Adsorption of Benzalkonium Chloride on Gauze Sponges* *

Temperature	Exposure Time	Concentration of Benzalkonium Chloride, Mg/Ml.		Quaternary Removed, Mg.	
		Original	Final	Per Sponge	Per Gram
25 C (room temperature)	10 min.	1.000	0.905	8.08	1.58
	30 min.	1.000	0.875	10.63	2.08
	1 hr.	1.000	0.885	9.78	1.91
	7 hr.	1.000	0.830	14.45	2.82
	26 hr.	1.000	0.785	18.28	3.57
	48 hr.	1.000	0.783	18.45	3.60
4 C	48 hr.	1.000	0.820	15.30	2.99

* Gauze sponges were 8×4 in., 12-ply (Johnson & Johnson). For this series, 10 sponges were immersed in 850 ml. of solution.



Graph 2 (drawn from Table 3).—Quaternary adsorbed by gauze sponges 8×4 in. as a function of time.

sorbed, depending on the concentration of compound in the fabric and also on the nature of the fabric. Wool and cotton, both containing 13.3 mg. per gram, lost 15% and 50% quaternary, respectively, when placed in distilled water.

To determine whether sufficient quaternary remains on sponges to give a bactericidal effect after rinsing them in water, the following experiment was conducted: Fifteen Zobec sponges were immersed in 250 ml. of a 1:1000 solution for 96 hours. The sponges were removed by means of a sterile forceps;

TABLE 4.—*Rate of Adsorption of Benzalkonium Chloride on Viscose Rayon Balls, Large* *

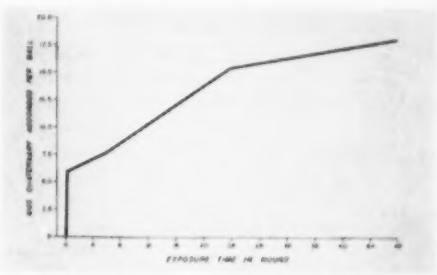
Temperature	Exposure Time	Concentration of Benzalkonium Chloride, Mg/Ml.		Quaternary Removed	
		Original	Final	Per Ball	Per Gram
25 C (room temperature)	10 min.	0.950	0.875	6.38	6.27
	30 min.	0.960	0.890	5.95	5.86
	1 hr.	0.960	0.920	5.95	5.86
	6 hr.	1.010	0.920	7.65	7.53
	24 hr.	0.995	0.815	15.30	15.06
	48 hr.	1.000	0.790	17.85	17.57
4 C	48 hr.	1.001	0.859	12.07	11.88

* Viscose rayon balls (Johnson & Johnson). For this series, 10 viscose rayon balls were immersed in 850 ml. of solution.

The effect of concentration can be seen in Table 5, where increasing concentration simultaneously increased substantivity. Table 6, where skin on the back of three subjects was used, also indicates a similar concentration effect.

It is significant that smaller amounts of quaternary are adsorbed as concentration decreases. Reuse of a solution therefore results in less quaternary removal per object. Thus, assuming that one starts with 1.256 mg. per milliliter, 10 sponges (8 by 4 in.) would remove 16.66 mg., resulting in 1.060 mg. per milliliter. At the lower concentration, an additional 10 sponges would (Graph 4, drawn from Table 5) remove 16.30 mg. per sponge. Thus, when one has reached a concentration of 0.2 mg. per milliliter, or 1:5000, only 5.5 mg. is removed per sponge.

Goldsmith and Latlief [7] have reported that adsorbed quaternary can also be desorbed. After one vigorous rinsing in 1000 ml. of sterile distilled water, an inoculum of 1×10^6 organisms (E. coli 26 ATCC) per sponge was destroyed within two minutes. After two vigorous rinsings, 0.14% remained at the end of four minutes, using the same inoculum. This represents a reduction of 99.86%. Desorption occurs

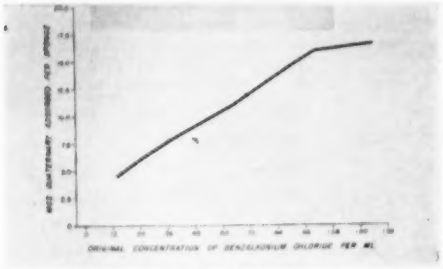


Graph 3 (drawn from Table 4).—Quaternary adsorbed by viscose rayon balls (large) as a function of time.

TABLE 5.—*Adsorption of Benzalkonium by Gauze Sponges from Solutions of Varying Concentrations* *

Concentration of Benzalkonium, Mg/Ml.		Quaternary Adsorbed per Sponge, Mg.	% Reduction
Original	Final		
1.256	1.060	16.66	15.61
1.008	0.819	16.07	18.75
0.887	0.716	14.54	19.28
0.777	0.626	12.84	19.43
0.643	0.514	10.97	20.06
0.390	0.296	7.99	24.10
0.263	0.185	6.63	29.66
0.135	0.080	4.68	40.74

* This Table indicates the amount of quaternary adsorbed by gauze sponges at a number of concentrations. Ten sponges were immersed in 850 ml. of solution for 41 hours.



Graph 4 (drawn from Table 5).—Adsorption of benzalkonium chloride by gauze sponges (8×4 in.) as a function of concentration (adsorption isotherm).

sufficiently to decrease germicidal activity, but the quaternary molecules are preferentially adsorbed to cotton fiber to the extent that vigorous rinsing does not completely elute them. Furthermore, this attachment does not preclude their participation in bactericidal activity. Lecithin and polysorbate 80 U. S. P. (Tween 80) in a buffered solution (pH 7.2) were used to inactivate the quaternary after the desired period of exposure.

The highest dilution permissible in benzalkonium chloride solution which gives an adequate margin of safety and yet affords a tangible lower limit for residual concentration can be set at 1:5000 dilution. For E. coli, a suspension of 5×10^7 is destroyed within five minutes at 27 C in 1:32,000 dilution in purified water U. S. P. Lawrence [8] reports that a 1:18,000 dilution will destroy Staphylococcus aureus within 10

TABLE 6.—*Quarternary Adsorbed by Skin of Back* *

	Benzalkonium Concentration, Mg/Ml.		Benzalkonium Adsorbed per Sq. Cm. Skin, Mg.
	Original	Final	
Subject A	1.293	1.251	0.034
	0.986	0.955	0.025
	0.812	0.785	0.022
	0.810	0.771	0.032
	0.616	0.594	0.018
	0.502	0.486	0.013
	0.260	0.248	0.010
Subject B	0.988	0.964	0.020
	0.510	0.487	0.019
Subject C	0.996	0.965	0.025
	0.664	0.645	0.016
	0.329	0.307	0.018

* Using a special brass template, 80 ml. of quaternary solution in varying concentrations was applied to 97.8 Sq. cm. of skin for a period of 30 minutes.

minutes at 20 C; a 1:27,000 dilution destroys E. coli within 10 minutes at 20 C, both in aqueous solutions. Dennis[9] states that a 1:30,000 dilution is adequate for S. aureus, and 1:7500 for Pseudomonas aeruginosa, at room temperature.

Thus, a 1:5000 dilution (0.2 mg. per milliliter) kills all known vegetative cells except tubercle bacilli within 10 minutes. Solutions weaker than this should be discarded.

Recommendations for the use of this quaternary should also include, therefore, a detailed list of the substantivity of articles commonly immersed in such solutions. In this way a superior germicide will be used with maximum efficiency, and with correspondingly greater understanding of the dynamics of its action.

Other interfering substances, such as metallic cations and organic material,[8,10,11] which have been reported as factors altering the rate of disinfection of quaternaries, are not discussed. This omission is not due to a lack of appreciation of these factors, but, rather, is occasioned by the belief that hospital solutions freshly prepared with purified water U. S. P. do not contain enough of these substances to present a problem.

Another factor which we have not investigated, but which has been reported by Fogh et al.,[12] is the adsorption of quaternary upon glassware. They have found that scratched glassware adsorbs more quaternary than new, unscratched glassware. Six scratched test tubes reduced the concentration of a 1:50,000 solution of cetylpyridinium chloride by 27%. These authors also reported that vigorous shaking resulted in foam formation, which lowered the concentration of the quaternary in the liquid phase by 9.8% with a concomitant increase of quaternary in concentration in the foam. They were, however, dealing with an extremely high dilution of the compound, 0.02 mg. per milliliter. The solution of quaternary that one uses for bactericidal purposes, 1:1000, 1 mg. per milliliter, is of high enough concentration that the adsorption by the vessel itself is negligible, unless it is badly scratched.

Summary

Time, temperature, and concentration have been found to be determining factors in adsorption of benzalkonium (Zephiran) chloride U. S. P. by objects immersed in such solutions.

It is recommended that original concentrations of this quaternary in use dilutions be made sufficiently great (1:750) to make allowances for adsorption upon immersed articles and leave continued bactericidal activity in the residual solution.

The high dilution 1:5000, which destroys all known vegetative cells except tubercle bacilli within 10 minutes, is recommended as the limiting residual dilution.

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Current Trends in Treatment of Tibial-Shaft Fractures

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Introduction and General Considerations

It is estimated that the American people sustain approximately 2,000,000 fractures yearly. These injuries, which include a significant number of tibial-shaft fractures, are treated by general practitioners and general surgeons, as well as orthopedists.[1] It is timely, then, that they be discussed at this Clinical Meeting. It is the purpose of this paper to report our 10-year experience, i. e., from 1946 to 1956, with 71 tibial-shaft fractures. The area involved is shown in Figure 1. Fractures of the knee and ankle are not included in this analysis. It has been said by some that it requires less skill to carry out an open reduction upon the tibia, and that for the occasional fracture surgeon it is prudent to follow the more radical form of treatment.[2] We are not in agreement with this concept, and we shall describe in some detail our method of closed management of these fractures and stress the favorable results that ordinarily accompany the less radical approach. Although we do not subscribe to the axiom that there is only one way to treat tibial-shaft fractures, we do feel that the degree of morbidity associated with open reductions upon this bone is perhaps greater than is generally realized.

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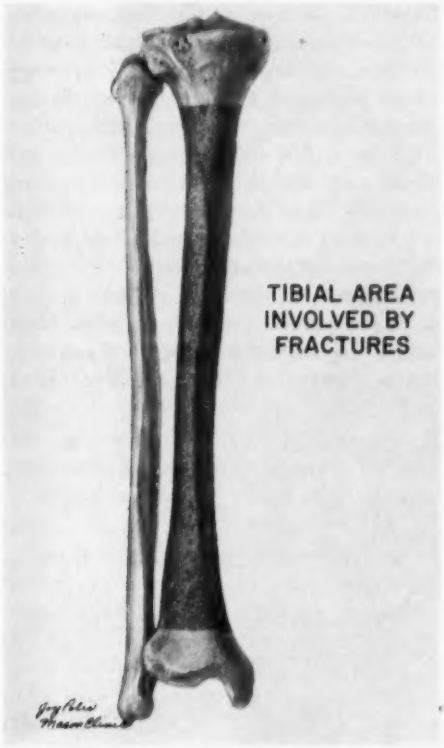
Fig. 1.—Shaded area depicts region of tibial diaphysis involved by fractures.



We have analyzed this series with particular reference to the healing time and the morbidity. The time of union was arbitrarily taken as that period from the day of fracture until the discontinuance of external support. This was usually the time when the final cast was removed, although in the few instances where a brace was also utilized during the final weeks the union time was extended to include this period. Complete bony consolidation was not always present at the time external support was discontinued, but there was x-ray evidence of early bony bridging and all the fractures were clinically solid.

Material

Of the 71 fractures that were available for analysis, 19 had occurred in children, i. e., in persons under 15 years of age. In this group, time of union varied from 4 to 11 weeks, with an

TABLE 1.—*Method of Treatment—Fifty-Two Adult Patients*

Method of Treatment	Simple	Compound	Total
Closed	27	7	34
Open	14	4	18
Total	41	11	52

average of 6 weeks. These were simple fractures in every child, and all were treated closed with a long leg cast. No external skeletal fixation was used in these patients. The clinical end-result was good in all, with no significant morbidity. We have had occasion to see a few children, treated elsewhere, who have had open methods carried out upon the tibia. In at least one instance in which screw fixation was utilized in an oblique fracture, occurring in a 9-year-old child, approximately 2.5 cm. of overgrowth occurred in a short period as a result of treatment. We feel, as do most surgeons, that there are few if any, indications for open operation in treatment of closed fractures of the tibia in the child.

The remaining 52 fractures of the tibia occurred in patients ranging from 16 to 85 years of age. The methods of treatment are shown in Table 1. In this adult group, 41 fractures were simple and 11 were compound fractures. A total of 34 of these fractures, made up of 27 simple and 7 compound, were treated by closed methods, while the remaining 18, of which 14 were simple and 4 compound, were treated by operation and some form of internal or external skeletal fixation.

Method of Treatment

We believe that individualization is extremely important here, as elsewhere. However, for the more or less routine spiral oblique and transverse fractures, our experience has shown that closed treatment carries with it less morbidity and at least as early, and in some instances an earlier, good result. Since the tibia is essentially a subcutaneous bone, particularly along its anteromedial aspect, the surrounding soft tissues are frequently damaged at the time of injury. Surgery in such areas may lead to wound diastasis, more particularly if closure is carried out under tension. Most infected tibial fractures have been preceded by wound derangement and breakdown. It is wise, then, to utilize methods of treatment in these fractures that avoid incisions over the fracture site whenever possible, and therefore closed reduction and plaster fixation remain for us the old stand-by of treatment. We do not entirely condemn the open treatment of tibial fractures, for we believe there will always be a place for internal fixation in certain compound and segmental fractures of this bone, especially when there is displacement of a large fragment. These are serious injuries, and the treatment of each patient must be carefully decided upon by the surgeon as he evaluates the patient, the lower extremity, and the x-rays of the tibia; then—and only then—can he formulate a plan of action and follow it through. The closed treatment is no panacea.

In our hands, we have found the following routine to work out very satisfactorily in the closed management of tibial fractures. The patient, who is brought either to our offices or to the emergency room of the hospital, has his limb immediately immobilized in a pillow splint, as shown in Figure 2. X-rays of the limb are made, either with the regular or with the portable machine, depending upon the circumstances of his admission, time of day, or other injuries. After an evaluation of the patient, with particular reference to his general health and other injuries, all things being favorable, he is given appropriate premedication and taken immediately to the operating room, where a sciatic- and femoral-block anesthetic is administered. After the usual skin preparation, a medium-sized Steinman pin is inserted transversely through the upper tibia in the region of the tibial tu-

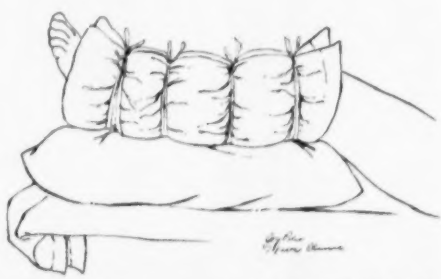


Fig. 2.—Lower extremity is immobilized in pillow splint and elevated on second pillow.

Fig. 3.—Drawings show completed cast with pin above fracture site in region of tibial tubercle incorporated in plaster, as well as flexion of knee.

bercle. The fracture is then manipulated and a long leg cast applied over adequate padding. We have found that if the cast is applied in sections, the surgeon or his assistant is better able to hold the limb in alignment and prevent displacement of the fractured fragments. It is frequently necessary to place the foot in moderate equinus position to prevent posterior angulation of the fracture at the time of application of the original cast. The pin is incorporated in the cast, and the knee is held in 20 to 25 degrees of flexion while the long leg cast is applied, as shown in Figure 3.[3] Check-up x-rays are obtained in two views, and the cast is wedged as indicated—immediately if undue angulation is present. The cast is frequently split anterolaterally over its distal third, allowing for easy spreading if necessary. The patient is then returned to his bed and the limb elevated with two or three pillows, or preferably suspended for several days in a sling from an overhead frame. Active toe exercises are encouraged from the start; these, together with elevation, keep the swelling at a minimum. The patient is ordinarily comfortable within a few days, at which time he is allowed up in a wheel chair, followed after 7 to 10 days by gradual ambulation on crutches. The pin in the upper tibia serves to anchor the cast, and we have found there is less tendency toward displacement of the fractured fragments when this simple fixative device is utilized. It adds tremendously to the patient's comfort as well. The original cast is usually not changed for six to eight weeks, although several x-ray checks of the fracture site are made during this period. The pin is withdrawn at the time of removal of the first cast, and the fracture is evaluated clinically, and by x-rays in two views out of plaster. A new long leg cast is ordinarily applied; occasionally a short leg cast will suffice. The next cast change occurs in four to six weeks, and in many instances the patient is then ready to begin weight bearing, in a skin-tight, short leg cast with a Stryker heel.

We occasionally have inserted a pin below the fracture site but feel this is unnecessary in the treatment of most tibial injuries. When the distal pin is placed either in the lower tibia or in the os calcis, it should be removed within three to four weeks, so that there will be no tendency for the fractured fragments to be held apart. Periods of distraction as short as eight days may delay union for many months, a serious hazard in either closed or open fractures.[4]

We have not found it necessary to utilize traction in our series but believe it has a definite place in difficult situations. The patient with a very swollen limb, particularly in some compound injuries where a cellulitis or other infection has occurred, makes the use of the Bohler-Braun frame with traction desirable in many instances. If this method is used, the application of a long leg cast at the earliest opportunity would be expedient and good treatment. The so-called traction cast, as described by Watson-Jones[5] and Hampton[6] in unstable fractures of the tibia, has merit in some instances, but we have not had occasion to use it. More recently, Hampton[6] and others[7] have advocated the closed reduction of certain fractures of the tibia, particularly

segmental fractures, with closed intramedullary nailing. We believe this method of treatment has definite indications and promise, but is not to be carried out by the occasional fracture surgeon.

We have treated puncture-wound fractures that are compounded from within out, with minute or small skin openings, by thorough cleansing of the wound and application of a sterile dressing, proceeding as with a simple or closed tibial fracture. These patients have in recent years been administered an appropriate wide-spectrum antibiotic for 10 to 14 days. None of them has become infected, and in general their course has been the same as with simple fractures. In tibial fractures that are compounded from without in we feel that the usual débridement and wound toilet should be carried out. We also advocate cultures of the wound at the time of débridement of open fractures.[8] We likewise make curved incisions in enlarging these wounds, and favor the generous use of relaxing incisions, with split-thickness skin grafting laterally as necessary.[8] The key to débridement is the removal of all foreign material and necrotic, or potentially necrotic, tissue. Tissue that floats as the wound is lavaged with saline should, of course, be excised. Under ordinary circumstances following wound closure, if it is appropriate, and it may or may not be, depending on the time element and the degree of contamination that is found in the wound, a Steinmann pin is inserted transversely through the upper tibia and a cast applied, maintaining good alignment of the fractured fragments. Simple forms of internal fixation, such as an oblique screw or intramedullary pins, may occasionally be utilized, but we have in general not favored internal fixative devices in the treatment of compound tibial fractures. The patient is covered with a wide-spectrum antibiotic, and booster doses of tetanus toxoid are administered to patients who have had previous immunization. Tetanus antitoxin U. S. P., 1500 units, is given to those who have not had previous tetanus toxoid and who demonstrate no sensitivity to horse serum. In the past, gas gangrene antitoxin in therepeutic doses has been administered in many of these cases of severely compound fractures, but recently it has been pointed out by MacDougall[9] that this biological is of no value prophylactically in the prevention of gas gangrene. He has advocated the usual thorough débridement and administration of 600,000 units of penicillin at 12-hour intervals, plus one of the antibiotics effective against Gram-negative bacilli. We should like to emphasize strongly that in severely contaminated compound fractures, even though the time element be short, the wound should be left open and secondary closure or grafting carried out at the appropriate time.

TABLE 2.—*Average Union Time—Fifty-Two Adult Patients*

Method of Treatment	No. of Cases	Average Union Time, Wk.
Closed	33, excluding 1 death	18
Open	14, excluding 4 nonunions	20

Analysis of Results

The patients that were treated were studied particularly from the standpoint of the time of union, morbidity, and complications. Of the 52 adult tibiae, 34 were treated closed, and these included 7 compound fractures of the puncture-wound type. Of the 34 adult tibiae treated by closed methods, the union time varied from 4 to 40 weeks, with an average of 18 weeks, and of the 18 tibiae treated by initial open surgery, 14 went on to union in from 15 to 38 weeks, with an average of 20 weeks, as shown in Table 2. Nonunion did not occur in the group treated by closed methods. It is significant that there was very little morbidity in the group undergoing closed treatment of their fractures. One 74-year-old man sustained a bowel obstruction associated with volvulus secondary to fractures of transverse processes, rather than to the tibial fracture. Two patients developed a moderate dermatitis beneath the cast, which

cleared rather promptly after removal of the plaster. The edema following cast removal was handled easily with supportive bandages, elastic stockings, and active use of the part. The oldest patient in this series, who was 85, died 23 days after fracture, of cardiorespiratory failure. This was the only death in the entire series.

The complications and morbidity in the two groups are shown in Table 3. In the group treated by surgery, four developed nonunion. The tibiae that failed to unite were equally divided between the simple and the compound fractures. Two patients required a year of immobilization after a second operation, while another required nearly two years of external support before union occurred. The fourth patient sustained a severely compound, comminuted fracture that was treated with two pins above and two pins below the fracture site, after débridement and wound closure. This person's wound healed primarily, but union failed to occur. He was transferred to another hospital and was still under treatment 14 months following his fracture. Here, again, two persons had a dermatitis of some significance associated with their cast. One required removal of a plate because of reaction about its screws, 10 months after fracture. One became infected. There were no deaths. Although the comparison is certainly not strictly a valid one, as many of the fractures treated openly were of the severer type, nonunion and the morbidity as a whole were nevertheless much more frequent in the patients treated with open surgery than those treated by closed methods.

Although we were at one time quite ardent advocates of open surgery, on certain spiral oblique fractures at the junction of the middle and the lower third of the tibia, our ideas have gradually changed in this regard. We now share the opinion of Urist et al.[10,11] that closed reduction is the procedure of choice in most instances. Patients whose fractured tibiae seem "ideally suited" to operative reduction are frequently equally as good or better candidates for closed methods. Patients with fractures that remain grossly displaced after one or more attempts at closed manipulation are still considered candidates for open surgery. Such fractures, however, are in the minority.

Summary and Conclusions

A 10-year experience in the treatment of 71 fractures of the shaft of the tibia is presented. We describe in some detail our closed management of fractures of the tibia. Open reduction of these fractures does not shorten the healing time or prevent nonunion. The importance of conservative treatment in fractures of the tibia is stressed. Open reduction of closed fractures of the tibia is seldom justified. We feel that the reëmphasis of closed methods in the treatment of fractures of the tibial shaft comes at a very important time, since resistant staphylococci are presenting an increasing problem in hospitals throughout the nation.

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TABLE 3.—*Complications and Morbidity—Fifty-Two Adult Patients*

Method of Treatment	No. of Cases	Nonunion	Infected	Misc.	Total	Per Cent
Closed	34	0	0	2	2	5.8
Open	18	4	1	3	8	44

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Case Reports

Traumatic Cyst of the Spleen

VIRGIL E. JEANS, M.D., Joplin, Mo.

Cysts of the spleen as a pathological entity were first recorded by Andral in 1829. The sparse, relevant literature since then attests to the fact that they are unusual, and a review of the literature by Bell, in 1951, revealed that a total of only 188 had been recorded.

The purpose of this report is to review briefly the pertinent literature and to present a case of post-traumatic splenic cyst, one of unusual size and presenting many problems in differential diagnosis.

Cystic tumors of the spleen may be classified as follows:

True cysts—lined by specific secreting membrane
- Epithelial
  1. Dermoid
  2. Epidermoid
- Endothelial
  1. Lymphangioma
  2. Hemangioma
  3. Polycystic disease
  4. Some serous cysts
- Parasitic—lined by protoplasmic matrix containing numerous nuclei
  1. Hydatid cyst caused by Echinococcus

False cysts—no specific secreting lining
- A. Hemorrhagic
- B. Serous
- C. Inflammatory
  1. Acute necrosis in infection
  2. Chronic tuberculosis
- D. Degenerative liquefaction of infarcted areas caused by embolism or arterial thrombosis.

It has been reported that 80% of splenic cysts are false. Usually they are large, solitary, unilocular, and hemorrhagic. Trauma is reported in slightly more than 50% of the cases. Little is known of the precise etiology, but the following case report suggests certain facts which may be significant.

Submitted for publication May 16, 1957.

Report of Case

A man aged 26 was admitted to St. John's Hospital, Joplin, Mo., on Jan. 7, 1947, complaining of weakness, anorexia, weight loss, and abdominal distention. The past history revealed that the patient had sustained severe abdominal trauma in an automobile accident on Nov. 2, 1946. Initial treatment was administered elsewhere. Physical examination revealed a huge abdomen which presented the classical signs of ascites. A gastrointestinal x-ray series tended to substantiate this finding by showing the stomach displaced to the left and the small bowel floating on free liquid. On Jan. 10, over 9000 cc. of dark-brown fluid was removed by paracentesis. Grossly, this fluid appeared to be bile-stained, but chemical examination revealed no bile. The specific gravity was 1.015. No untoward reaction followed the paracentesis, and the patient was discharged from the hospital improved.

On Jan. 21, nine days after his dismissal from the hospital, he was again admitted for evaluation. There had been a rapid reaccumulation of fluid, and his complaints and findings were essentially as they had been on first admission. This time 3000 cc. of similar fluid was removed by paracentesis. He experienced relief and was discharged from the hospital. On Feb. 2, the patient was again admitted to the hospital with the same complaints. This time the abdominal swelling was taking on a definite shape. It had the appearance of a large football, occupying the entire left side of the abdomen. The percussion note was dull over this area and there was a fluid wave, but it did not shift with movement. It was then apparent that he had a traumatic cyst, probably caused by laceration or rupture of the spleen.

Exploratory laparotomy was done through a transverse incision on Feb. 7. A large, thick-walled cyst, measuring 45×18 cm., was encountered. It extended from the diaphragm down into the pelvis. There was constriction at the costal margin and also at the brim of the pelvis, making the cyst look like three separate pouches. The spleen was attached to the upper portion. The cyst was partially collapsed by removing 8000 cc. of dark-brown fluid. After this, the cyst and spleen were removed as one

specimen. The spleen itself was enlarged two or three times its normal size, and its capsule was continuous with the capsule of the cyst. The cyst wall was friable, brownish-green in color, and shaggy in appearance. Microscopic study revealed it to be fibrous tissue with attached blood clot. The operation was well tolerated by the patient, and his convalescence was uneventful.

Comment

This case presented several interesting problems, the first being the diagnosis. Initially, the patient was thought to have ascites, but it was difficult to rationalize this diagnosis. There was no evidence of cardiac decompensation and no diagnostic signs of portal hypertension. The excess fluid was confined to the abdomen, but it was inconceivable that even a large laceration of the liver could result in cirrhosis so quickly. There was no bile in the aspirated fluid, a finding which was further argument against liver damage as the primary injury. Furthermore, transudation following injury could account neither for the amount nor for the persistence of the fluid. It was not until the cyst wall had become thick enough to take on a definite shape that the correct diagnosis was suspected.

The second, and more interesting, problem was the mechanism for the formation of the fluid in the cyst. Microscopic examination showed that the cyst wall was fibrous tissue. This could have been the split capsule of the spleen, which had expanded as hemorrhage progressed. This supposition seemed unlikely, because the initial profound shock indicated an acute severe hemorrhage and the thin splenic capsule could scarcely dilate to contain over 9000 cc. of fluid. Preoperatively, the rapid accumulation of fluid, after repeated aspirations, suggested that the cyst wall was a secreting membrane, but this origin was not substantiated by microscopic study.

A more logical explanation is that there was a tremendous hemorrhage from the spleen at the time of injury; the clot became organized as he recovered from shock, and it took on a definite capsule. This capsule was continuous with the splenic capsule. As the blood clot decomposed and liquefied in the center, osmotic pressure caused it to draw in fluid from the surrounding tissues. This chain of events is the same as the neurosurgeon sees in the patient with chronic subdural hematoma; that is, a patient receives a blow to the head and appears to have sustained a trivial injury. It is only later, perhaps two or three months, that he begins to have localizing signs and symptoms of intracranial pressure. The clot, which of itself was not large enough to produce symptoms, increases in size as more fluid is drawn into it by osmotic pressure, and thus produces the typical chronic subdural hematoma syndrome. I believe the same thing happened in this case. The clot, which was large because of the severe shock initially, was the nidus for the formation of a huge cyst. The aspirations relieved the intracystic tension and increased the concentration of the residual contents, thus enhancing the osmotic pressure and thereby causing rapid refilling of the cyst.

Summary

A case of traumatic cyst of the spleen is presented.

Rupture of the spleen with marked hemorrhage is not necessarily fatal.

Certain problems in differential diagnosis are indicated.

Mechanisms for formation of cysts are suggested.

The treatment is surgery.

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THE INDEX

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SHOULDER ARTHRODESIS:
1. Radiograph (A-P view). Shoulder prior to surgery. Note atrophy of muscle and soft tissue, and relaxation of joint.

2. Skin draped. Saber incision outlined at acromion.

3. Acromion denuded of cartilage. Shoulder capsule opened.

4. Acromial process of the scapula osteotomized.
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5. In succession: Flap is osteotomized in head of humerus; cartilage is denuded from head of humerus, which is joined to acromial process.

6. Acromial process now in head of humerus.

7. Cuff of shoulder capsule reefed to scapula.

8. Wound closed and prefitted cast applied.

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